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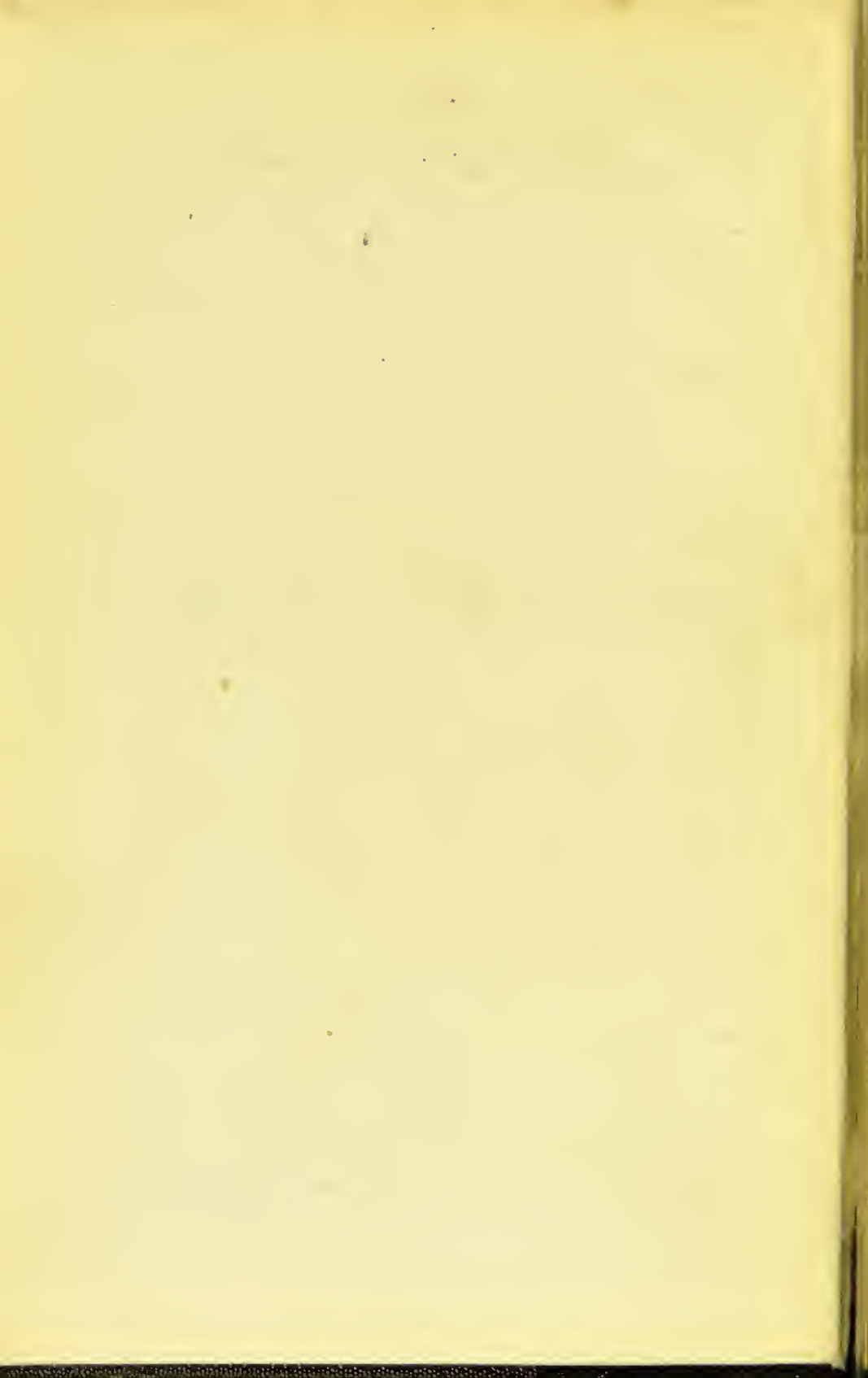
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THE
PRACTICE AND THEORY OF MEDICINE
VOL. II.



UNIVERSITY OF LEEDS

A MANUAL OF
THE
PRACTICE AND THEORY
OF
M E D I C I N E

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LUPUS

Under the term 'lupus' two distinct skin-affectiions are included, having little in common—*i.e.* *L. erythematosus* and *L. vulgaris*.

LUPUS ERYTHEMATOSUS

This is the result of a peculiar inflammatory process causing a cellular infiltration which eventuates in atrophy of the affected skin, accompanied by scarring.

Etiology and Pathology.—The disease is much more common in women than in men, the proportion being two or three to one. It commences in adult or early middle-life, being very rare in childhood and old age; it is more common in cold climates; chilblains often occur in those suffering from it. Though its etiology is obscure, the generally accepted theory is that the inflammatory process originates in some local or constitutional disturbance of the circulation, in which the bacillus of tubercle plays no part, though its association with tubercle is still maintained by French dermatologists, who regard the inflammatory process as one produced by the toxins of tubercle.

Microscopic examination reveals the inflammatory process as originating in the vascular walls and infiltrating the skin with great numbers of small round cells. The hair-follicles, sebaceous and sweat glands, participate in the process, and as the cell-growth degenerates atrophy supervenes, which produces the characteristic atrophic scarring, which is seldom or never associated with ulceration.

Symptoms.—The disease affects the face, head, hands, toes, and ears most commonly, but it may involve the skin over any part of the body. The most characteristic site or distribution is that in which two large, more or less symmetrical, patches form upon the cheeks; these are connected together by a transverse bridge over the nose, the whole having some resemblance to the outspread wings and body of a butterfly or bat.

Two well-marked varieties are seen—*i.e.* the *discoid* and the *disseminate*. The discoid form commences in small red spots which cluster or remain discrete, and usually have a symmetrical distribution on the cheeks, nose, ear-tips, lips, scalp, hands, or feet, and sometimes on the neck. The spots fade on pressure, have a red raised border, while the depressed centre is occupied by a small yellowish crust or scale

consisting of a plug of sebum, which extends into the orifice of the dilated sebaceous duct, or fits into depressions in the skin. These 'primary eruptive spots,' at first not larger than one-sixth of an inch in diameter, slowly extend at the circumference, and finally form variably sized symmetrical patches.

In the disseminate variety, numerous crops of spots coalesce to form patches of large dimensions, which may involve a considerable proportion of the cutaneous surface. The patches are bounded by a more or less sharply defined, raised, irregular border, which is hyperæmic, the central portions being lighter in colour and somewhat depressed and scaly. Comedones are frequently present about the margins of the patch, especially when the nasal wings are the site, and by some authorities this sub-variety has been styled *sebaceous* or *seborrhœic lupus*. The central scaly depressed part fades, and gradually presents the characteristic whitish atrophic scarring without passing through any ulcerative stage. When the scalp is the seat of the disease the hairs are shed, and permanent baldness may result.

Where the region affected is the skin of the fingers or toes, the disease is of a more distinctly erythematous type. The scaliness or dried sebaceous secretion being absent, the condition becomes apparently identical with chilblains, and hence it has been described as *chilblain lupus*. A similar appearance may be observed when the disease affects the ears.

The *telangiectic* form of erythematous lupus of Crocker, in which the patches are free from scaliness and resemble the circus clown's red daubs on the cheeks, is caused, according to this authority, by dilatation of the small blood-vessels. Crocker also describes a rare *nodular* form of the disease which may closely resemble lupus vulgaris.

The affection may in some instances select the mucous membrane of the nose, mouth, throat, and larynx, but when this occurs generally the skin has been primarily affected. Smith recently has published the results of an investigation of fifty-six cases in which the mucous surface was minutely examined, and every fourth case showed involvement by the disease as demonstrated by the presence of reddish violet-coloured spots, which finally ended in small dense white plaques with converging striæ.

The course of the disease is essentially chronic and progressive, with variable periods of quiescence; some cases, after lasting for twenty or more years, permanently cease to exhibit any activity, no evidence of the disease remaining save the atrophic scarring. Often mild types of the affection spontaneously recover, leaving scarring which is almost invisible. In the disseminate type, however, the disease

may sometimes be witnessed as commencing in an acute manner, or as taking on an active or eruptive character, in which the inflammatory process may become as formidable as in erysipelas, and from which death has been known to result. Usually there is no constitutional disturbance, and local symptoms are generally so slight as to cause little discomfort, except in the acutely erythematous types, when there may be some itching, tingling, and pain, as in chilblains.

Diagnosis.—When upon the face, the butterfly-distribution is not liable to be mistaken for that of any other skin-disease ; but it must be remembered that it usually takes some years for the disease to travel over the cheeks and across the nose. The depressed central whitish scaly cicatrix, surrounded by an erythematous raised border, with plugging of the sebaceous ducts, readily distinguish it from every other skin-affection on the face, nose, or ears. Upon the scalp, the baldness which always follows may be mistaken for that of *alopecia areata*, from which it is differentiated by the presence of the raised erythematous margin. *Favus* and *ringworm* may also be simulated, but the absence of the fungi or spores of these diseases settles the diagnosis.

Chilblains on the toes and fingers may so closely resemble *L. erythematosus* that the diagnosis may have to be postponed till the advent of warm weather, which always clears up the simple chilblain-lesion.

Lupus vulgaris is differentiated by the following characteristics—viz. the age of the patient, who is nearly always under puberty when the disease commences ; the presence of apple-jelly nodules ; of ulceration, which may be said never to occur in *L. erythematosus* ; and of the tendency of the true lupus to extend deeply below the cutaneous surface, destroying cartilages and causing shedding of bones.

Rosacea is differentiated by the presence of pustules and by the absence of the fine crusts or scales with the sharp plugs projecting from their deep surface.

Prognosis.—In the acute disseminate form death may occur, but such cases are rare in Great Britain. The localised forms are prone to improve under treatment, or spontaneously to resolve, in proportion to the preponderance of the erythematous element as contrasted with the seborrhœic.

As regards the duration of the disease and the extent of atrophic scarring liable to be left permanently behind, it is almost impossible to forecast till the results of treatment have been watched for some time.

Treatment.—Internal medication is most unsatisfactory. The following drugs have been supposed to exert a beneficial action—i.e. arsenic, phosphorus, iodides, quinine, salicin, and ichthyol. Cod-liver oil is the most reliable agent, in

conjunction with measures directed to the correction of any obvious departure from the standard of health.

Local treatment is also unsatisfactory and empirical. In the acute erythematous form the drugs indicated in the early stages of eczema should be resorted to, irritating applications being productive of harm. Mild lead and zinc ointments or lotions should be employed till the hyperæmia is subdued somewhat, after which ichthyol is often beneficial.

Where much scaliness or crusting exists the treatment suitable in inveterate chronic eczema may be tried, as soap-solution, followed by tarry compounds. Painting the patch over repeatedly with collodion diminishes the hyperæmia by causing contraction of the small vessels.

Caustics, as pyrogallic and salicylic acids, sodium ethylate, strong solution of pernitrate of mercury, curetting, linear scarification, and the thermo-cautery have all at times been followed by benefit.

The Röntgen-rays, Finsen's light-treatment, and radium-therapy may be tried, but they are less successful than in lupus vulgaris. Recently excellent results have been obtained by high-frequency currents, which should be tried in all chronic cases.

LUPUS VULGARIS

This is a skin-affection produced by the presence of the tubercle bacillus, which causes by its irritation a small-celled growth, starting in the deep part of the corium, finally infiltrating all the layers of the skin, and tending towards ulceration or atrophic changes through the degeneration of the nodules caused by the parasite. It is often described as Lupus exedens, owing to the frequency with which ulceration occurs.

Etiology.—Age and sex are marked predisposing factors, the disease being at least two or three times more frequent in females, who are nearly always attacked before puberty. A family history of tuberculosis is common, though many of the patients are apparently in good health, and only a small proportion become phthisical. The disease sometimes follows measles and other fevers which lower the vital resistance to the bacillus; and its distribution on the body would suggest that exposure to cold acts as a predisposing cause, since it is comparatively rare on regions usually well covered.

Direct inoculation by the bacillus has been proved in many instances, as in the operation of circumcision, in piercing the lobes of the ear for earrings, in the performance of ordinary vaccination, and following tattooing and other wounds.

Indirect inoculation has been witnessed where the disease has invaded the skin from underlying tubercular glands or from remote foci; and some authorities believe that it may be even dependent upon infection caused *in utero*. Probably

in every case there is the impaired natural resistance to the bacillus, which is believed to be handed down by heredity to those who succumb to phthisis and other forms of tuberculosis, though lupus cannot be said to be hereditary. In a very few cases the disease has been regarded, as originating in contagion, the patient having been long in close contact with a lupus victim.

Pathology and Pathological Anatomy.—As already stated, the pathology of lupus is simply that of a local tuberculosis of the skin; but clinically it differs materially from scrofulodermia and verruca necrogenica, and even from lupus verrucosus, though all these are the result of the invasion of the skin by the tubercle bacillus.

(The so-called 'Tuberculides'—*i.e.* *lichen scrofulosum*, *acne* and *eczema scrofulosum*—are supposed to be the result of the inflammation produced by the toxins of the bacillus, though the bacilli themselves are not present in the lesions.)

The nodules of lupus may be regarded as identical with the tubercular nodules found in other tissues, but, owing to the difficulty which the parasite encounters in its growth in the corium, the bacilli are very few in number and are usually found with difficulty. The marked local reaction which follows tuberculin-injections demonstrates the nature of the disease, even when no bacilli can be found in the tissue or cells scraped from a lupus-patch. The typical nodule of lupus is a granulomatous structure composed of giant-cells, and a fibrous stroma infiltrated with epithelioid and small round lymphoid cells. Often even in a series of slides only a solitary bacillus may be visible in a giant-cell. The nodule ultimately tends to degenerate after it has grown to the extent of displacing the fibrous elements of the skin and reached the surface, though still covered by the translucent epithelium. When the degenerative change eventuates in softening, necrosis, or ulceration, the typical *lupus exedens* is the result; but the degeneration may show itself in a fatty or fibroid change without ulceration, in which case a fibrous cicatrix results, when the clinical picture is described as *lupus non-exedens*. In either case the epithelium of the sebaceous and sweat glands and of the hair-follicles are involved and destroyed in the irritative process started by the bacillus.

Symptoms.—The disease usually shows itself during childhood on the nose or cheeks, though no part of the skin is exempt. The characteristic feature is the presence of a papule, which is situated in the deep layer of the skin, and gradually becomes visible as it approaches the surface, being about the size of a pin's-head, and of a dull yellowish-red, which does not disappear on pressure. The papule increases in size, and gradually becomes a nodule, projecting above the level of the skin and covered by the translucent epidermal

layer. It has been likened at this stage by Hutchinson to a speck of apple-jelly under the epidermis. If the skin be covered by a piece of glass and pressure be applied, the nodule shows as a pale yellow spot, which at once differentiates it from all inflammatory lesions accompanied by the production of papules, as these latter would become invisible by this simple procedure.

Neighbouring nodules coalesce so as to form variously sized patches, around whose margins new papules or nodules from time to time appear, so that after many months or years a considerable area of the skin over the nose or cheek becomes thickened, red, and raised above the general surface. The margins of such reddened patches are firm and irregular or nodular, whilst the centres are soft and elastic, and the entire surface is covered over with fine scales, through which apple-jelly nodules may be visible studded over the red background.

Involution may occur in the centre of the patch, which is then replaced by a firm, smooth, burn-like atrophic scar; whilst the disease may continue to steadily spread by the formation of new nodules at its periphery. As a rule, long periods of quiescence occur, the disease being of an essentially chronic nature; but under injury, local irritation, or injudicious treatment quiescent patches may be stimulated to active growth.

Ulceration is more common than involution, the necrotic process destroying sometimes the entire depth of the skin involved in the patch, gradually eating away the nasal cartilages, and causing loosening or shedding of the nasal bones, though the osseous tissue is never directly invaded by the process. It is not an unusual experience to see spontaneous resolution occurring in one part of a patch, whilst at another active necrosis is steadily involving all the structures of the skin. When the disease reaches the mucous surface, its progress may become somewhat accelerated, and it may extend from the gums to the palate, and even to the larynx.

Commonly the disease spreads from a single focus. When more than one centre is present, they are never symmetrical, though sometimes a symmetrical distribution may be simulated when the disease begins in a focus situated upon the bridge or tip of the nose and extends uniformly in the direction of each cheek.

There is a rare form of lupus, described as *L. disseminatus*, which, starting in many different foci, may gradually invade a large extent of the entire cutaneous covering of the body. Though, as already stated, lupus is more common upon the face, it has been often found on the limbs and trunk, but scarcely ever is the scalp affected. It is rare on the middle of the forehead and upper eyelids, the palms and soles, neck and genitals. Though it may start primarily in the mucous

membrane of the nose, mouth, eye, or larynx, more commonly these regions are invaded by extension ; but the number of cases in which the disease starts in the mucous membrane just inside the nose is larger than most authorities realise. In one robust patient under the writer's observation, disfigured in youth by extensive lupus, which had become permanently cured, death resulted, long after middle-life, from tuberculosis of the bladder, which possibly may have been a lupus of the mucous surface ; tubercle bacilli were detected in small numbers in the urine.

The appearances left after the healing of the lupus-ulcers may be as varied as the manifestations of the disease in the spreading stage, but as a rule the necrotic process is superficial, though at times it may extend deeply, leaving large, deep, seamy scars. Upon the limbs, these may cause permanent contraction of joints, the skin being directly adherent to the fasciæ and bones, which had become stripped in the suppurative process which supervened upon the ulceration of lupus-patches.

A hypertrophic form of lupus in which there is solid lymphatic œdema simulating elephantiasis, and a fibroid variety in which the irritative process leads to the proliferation of much fibrous material, and a papillomatous variety, have been described ; but the essential pathological factor in all cases is the tubercle bacillus.

Erysipelas has been observed to supervene upon lupus, and sometimes the result has been most favourable to the patient, complete cure of the lupus having been occasionally recorded. In rare cases, spontaneous recovery has been known to occur. As a rule, the duration of lupus extends over many years—a quarter of a century being no unusual period of duration, if we include the quiescent periods or stages. As the patient advances in years the progress of the changes becomes much slower than those witnessed in the patches and nodules during childhood.

The symptoms proper of the disease are trifling. There is but little pain or itching, and in the majority of cases the general health suffers but slightly, or not at all. Ultimately, a minority of the cases—the exact percentage being very variously estimated by different authorities—show signs of phthisis. In a few, where the neighbouring lymphatic glands become infected and caseate or suppurate, death may result from wasting and hectic. In a very small percentage of cases epithelial cancer has been observed to attack lupus-patients in the scar-tissue long after healing.

Diagnosis.—The main characteristic feature upon which the diagnosis must rest is the apple-jelly appearance of the specific nodule. In some rare cases, where the irritative process caused by the bacillus only shows itself by a diffuse

infiltration, the diagnosis can only be made after the characteristic scarring has occurred ; but even in these cases, during some part of their progress, the specific nodules will be found in the margins of the patches if perseveringly sought after.

Syphilitic skin-lesions appear only after adult-life. There is a history of infection and of secondary symptoms ; the ulcerative process is deep and rapid, directly involving the bones ; the edges of the ulcers are sharp ; abundant dark foul-smelling crusts and offensive secretion accompany the syphilitic ulceration.

Scrofuloderma, which is but another clinical type of a tubercular invasion of the skin, is nearly always associated with evidence of caseating glands, lymphatic sinuses, or scar-tissue, though the lupus-nodule is not present, unless in those cases where both affections co-exist, as they not rarely do in typically strumous or scrofulous subjects.

From the general pathological and therapeutical standpoint, scrofuloderma and lupus vulgaris may be regarded as but different phases of the tubercular process. Both react locally to injections of the original tuberculin of Koch ; but it should also be remembered that leprosy does likewise.

Rodent ulcer occurs only late in life, eats deeply into the tissues, has an indurated margin and smooth centre ; no apple-jelly nodules are visible even after being pressed under glass, and it does not react to the old tuberculin-injections.

L. erythematosus is differentiated by the diagnostic points already mentioned upon p. 935, chief of which are the later age at which it first appears, the symmetrical character of the patches, the absence of ulceration and of the characteristic nodules, the involvement of the sebaceous glands, and the escape of the nasal cartilages.

Epithelioma begins after middle-life, and is accompanied with pain. Its site is fairly characteristic ; there is marked tendency to early implication of the lymphatic glands at the chin or angle of the jaw. The ulceration is deeper and more rapid, the edges are much indurated, and the surface granular or warty.

Eczema and *psoriasis* can only be mistaken for the rare form of disseminated lupus, which may be distinctly scaly, but there is no scarring and comparatively little infiltration, and the characteristic nodules and ulceration are absent in both eczema and psoriasis. Moreover, as pointed out by Malcolm Morris, in the disseminated variety of lupus there will always be found somewhere one or more of the patches which will present the typical characters of lupus.

Prognosis.—The disease is to be regarded as of a most chronic and inveterate nature ; though spontaneous healing is constantly to be seen in some small area of the patch, the tendency is towards certain necrosis or degeneration of the

tissue, unless active treatment be resorted to. There is little danger to life, except through some form of visceral tuberculosis, which supervenes in but a very small number of cases. The prognosis, as regards rapidity of progress and destruction of tissue, is graver in the very young, and the hope of a permanent cure after operative interference brightens in proportion to the advance in the patient's age, the tendency to recurrence being very great in children.

The prospects of permanent cure depend largely upon the amount of skin involved, being obviously much better where the patch is solitary and of small dimensions. The outlook is very unfavourable when the disease involves the mucous membrane to any extent.

Treatment.—The treatment of lupus is a large subject which the author has dealt with in detail in the fourth edition of his 'Dictionary of Treatment,' pp. 536 to 544. Only the general principles can be here mentioned.

Of internal remedies, thyroid feeding and injections of Koch's tuberculin are the only agents capable of exercising any beneficial action, and as a rule they are worthless unless when combined with local treatment.

The object aimed at in local treatment should be the entire removal of the diseased area of skin by excision, curetting or erosion, caustics, the cautery, electrolysis, multiple scarification, or preferably by a combination of these agents.

Photo-therapy by Finsen's method, in which solar or electric light is used to effect destruction of the tubercle bacilli, and radio-therapy or the use of the Röntgen-rays, and exposure of the lupoid tissue to the emanations from radium, have all given excellent results where the patient has been in a position to devote months to the treatment. The obvious advantages of these latter agents lie in the nature of the cicatrix which results; when successful, the deformity is trifling. The same remark applies to the results sometimes attained by high-frequency currents. These agents can sometimes be advantageously combined with erosion or caustic methods, which expedite their action. No known treatment will prevent the liability to recurrences. In a case seen recently by the writer the lupoid tissue was entirely removed, and a patch of healthy skin transplanted from the centre of the forehead to the bridge and sides of the nose. After the apparent success of this procedure, carried out in the most radical manner, the disease recurred in the transplanted skin. In another case, under Dr. Rankin's care, the most conscientious perseverance with erosion, Finsen's photo-therapy, Röntgen-rays, and other methods failed, till a few injections of tuberculin were followed by rapid healing. The influence of these latter was demonstrated by the simultaneous disappearance of an enlarged axillary gland within a

few days after the second injection. The cases which do best under photo-therapy are those with a high opsonic index. Those with a low index require in addition tuberculin injections.

LUPUS VERRUCOSUS

This so-called 'warty lupus,' though practically identical with or constituting a mere variety of lupus vulgaris, presents such a totally different appearance that it is here referred to under a separate sub-heading. It is a local tuberculosis of the skin appearing early in life; in the writer's experience all the cases were in males, but this was probably accidental. The hands and feet are most commonly the site of the disease, the face being very rarely affected. It may be regarded as identical with those cases of *verruca necrogenica* in *post-mortem* porters and slaughter-house operatives, where warty growth sets in about the spot where tubercle bacilli have been accidentally inoculated, as about the knuckles.

The appearance presented by a typical patch of the disease is that of a large, diffuse, flat wart, which appears to be very considerably raised above the level of the surrounding skin, owing to its being capped by a dense closely adherent horny crust, which often covers the entire surface of the growth. Fissures from which a bloody purulent secretion oozes are not unusually present; in some places at the margins beyond the crust the enlarged papillæ may be visible, but the apple-jelly nodules of lupus vulgaris are absent. The disease is very chronic, and tends to spread slowly at the periphery, though sometimes it may begin in neighbouring foci which coalesce.

In one typical case under the writer's care the warty growths had surrounded the lower part of both legs above the ankles, and they had existed for nearly thirty years. Sometimes in scrofuloderma the same warty formation may be observed, and ordinary lupus at some spots may exhibit the verrucose character, especially when the hands and feet are involved.

After the removal of the horny cap, the appearance is so characteristic that once seen it is afterwards easily recognised. The red weeping papillæ which constitute the moist infiltrated plaque distinguish the disease readily from *psoriasis*, with which, however, it is not likely to be confounded.

The disease, though of an extremely chronic and inveterate character, will yield to persistent treatment when the patient gives himself up to it and does not leave off before the complete destruction of the growth has been effected. The best plan is to commence with a few large poultices, after which the plaque is to be kept moist with salicylic acid made into a paste with glycerin. After a time, the process is expedited by curetting or the cautery, or by the application of strong caustics, of which Crocker prefers the fuming acid nitrate of mercury.

LYMPHADENITIS. (See under MEDIASTINUM,
 MESENTERIC GLANDS, TUBERCULOSIS, &c.)

LYMPHADENOMA. (See under HODGKIN'S DISEASE.)

LYMPH-SCROTUM. (See under CHYLURIA.)

LYMPHATIC LEUCHÆMIA

This condition is described under 'Leuchæmia.'

LYMPHATISM

This condition is also known as Status Lymphaticus.

Pathology and Etiology.—The disease is one in which the entire lymphatic system, glands, and vessels, thymus, spleen, intestinal follicles, bone-marrow, and tonsils become the seat of hyperplasia. Usually the superficial lymphatic glands are but slightly, if at all, involved, the main and most obvious changes being only recognised after death in the enlargement of the solitary and agminated intestinal glands, as well as those of the bronchi and mediastinum. The spleen is hypertrophied, and the thymus is usually also persistent, enlarged, softened, and swollen.

Nothing is known of its etiology, save that it has been often found associated with rickets, though it may affect children and young adults beyond the rachitic period. German authorities hold that the lymphatic hyperplasia is usually associated with the opposite condition—hypoplasia in other organs.

Symptoms.—The condition is often never suspected till the autopsy reveals the characteristic lesions, all symptoms being entirely absent. This association of the condition with the hypoplasia of the heart and vessels is maintained to be the explanation of many sudden deaths which occur in flabby, anæmic, rickety children, and in those suffering from adenoids. The condition, never suspected during life, has been repeatedly found upon *post-mortem* examination in children who have suddenly expired without any obvious cause during convalescence from feverish affections, diphtheria, during anæsthesia, or whilst bathing, and, in one well-known case, after a preventive injection of the anti-diphtheritic serum.

Diagnosis.—The diagnosis of the condition is most difficult, especially in the absence of enlargement of the superficial lymphatic glands; but mesenteric or mediastinal swellings,

when associated with adenoids, tetany, or laryngismus, should arouse suspicion of the underlying state. The blood-examination does not assist, the only change observable being a more or less marked leucocytosis, in which lymphocytes preponderate.

Lymphatism is intimately associated with the condition described as '*Thymus Tod*.' Some authorities regard the enlargement of the thymus-gland, which constitutes this affection, as but a part of the general status lymphaticus, which is confined to or most obviously seen only in the thymus. Death, in some cases, occurs from the pressure of the enlarged gland upon the air-passages, heart, or aorta, as cyanosis following inspiratory stridor and dyspnœa has been observed—a group of symptoms sometimes described as *thymic asthma*. Others consider the cause of death in such cases to be due to spasm not unlike, if not identical with, that seen in laryngismus stridulus; whilst the startling suddenness with which the fatal issue is sometimes associated resembles the swift cardiac paralysis or heart-block occasionally witnessed during diphtheritic convalescence.

In a still smaller percentage of cases of death from lymphatism, or thymus tod, in young adults, there may be a prolonged period of coma just before the final issue, as in diabetes, which is suggestive of a toxæmic explanation.

Treatment.—No known agents have any influence over the hyperplasia of the lymphatic system or enlargement of the thymus, even if the suspicion of its presence has been aroused. In some instances, where the thymus has been seen to be enlarged, and dyspnœa has supervened, life has been saved by tracheotomy and the passage of a long tube downwards through the tracheal opening, the thymus-tumour being afterwards elevated and attached to the cervical fascia.

In very young subjects the obvious indication for treatment is that of the rachitic condition, with which lymphatism is so often associated.

MALARIA

This is an infectious disease, characterised by fever of an intermittent, remittent, or continuous type, associated with anæmia and splenic enlargement, and caused by the invasion of the red blood-discs by certain protozoan parasites which are conveyed from man to man through the agency of the mosquito (*Anopheles*), which acts as their definitive host.

Distribution.—Malarial fever, often termed ague and intermittent fever, and known also by various titles indicative of the type of the pyrexia, has a wide distribution. Formerly common in the marshy districts in England and northern Europe, its area is being gradually restricted by improved sanitation and land drainage, so that its range in Europe

is practically confined to Southern Russia, Italy, and the countries bordering upon the Mediterranean, though still it may occasionally be met with, further north, in Denmark, the Rhine Valley, and along the Baltic shores.

The European type is, as a rule, mild, except in the malignant form, which occurs in summer or autumn on the Roman Campagna and in Greece.

In Asia and Africa the distribution of malaria is wide, and its type often most severe or malignant, especially in tropical Africa and along the Gold Coast. It flourishes in the low-lying lands in the vicinity of the great rivers of India and the marshy districts of Burmah, and most tropical regions, which supply suitable breeding-grounds for the mosquito; whilst it is comparatively rare in high altitudes. It is very prevalent in Central and South America; whilst the British possessions of North America are almost exempt. The disease, formerly very prevalent in the United States, is now chiefly confined to the regions about the Mexican Gulf. Australia, Tasmania, and New Zealand are practically as free from malaria as are the British Isles.

Etiology.—In considering the geographical distribution of malaria, it will be obvious that this must correspond to the distribution of the mosquito (*Anopheles*), by which the disease is spread. A warm or hot temperature is necessary for the multiplication of the insect, so that in temperate climates malaria is rare, except in the hot months at the end of the summer or autumn; whilst in the tropics the disease is more prevalent and severe in the spring and autumn, owing mainly to the facilities afforded to the insects for propagation during the periods of the rainy seasons in different localities.

Low-lying districts, in which surface-water is abundant, afford the best breeding-grounds for *Anopheles*, and, since the insect does not fly far from its native haunts, elevated regions, close by, may be quite free from malaria; as a rule, in proportion to the elevation the prevalence of the disease becomes rarer.

This is well seen in the Roman Campagna, where many natives entirely escape by sleeping in erections constructed at the tops of vertical posts. Even in cities where malaria is endemic, those who occupy the uppermost storeys seldom suffer, whilst the ground flats are malarious.

In well-drained localities, the absence of pools in which the larva can develop is sufficient to afford protection. The planting of marshy districts by eucalyptus and other trees acts in the same way as improved drainage.

The natives of malarial districts enjoy a certain degree of immunity, which is believed to be 'acquired,' since the parasites are found in the blood of an overwhelming proportion of the young children in all malarious localities. No age is,

however, exempt, and all races are liable to the disease ; but the white races certainly are more susceptible than the negro.

Two etiological facts, placed within recent years beyond the possibility of dispute, are that malarial fever is caused by a parasite, and that this is only communicated from man to man by the bite of the mosquitos of the genus *Anopheles*.

Laveran first demonstrated the parasite in 1880. Binz, with remarkable foresight, many years before had arrived at the conviction that the origin of the disease would probably be found to be a protozoan organism, being led to this view from observing the specific lethal action of quinine-solution upon certain infusoria abounding in hay-infusion.

The organisms (for the different types of malarial fever are now held to be the results of the invasion of different species of parasite) belong to the class of the sporozoa family—*Hæmamoebidæ*. They are referred to also as *hæmocytozoa*, and in the descriptions of the growth and multiplication of the parasite in the human red blood-discs it is frequently styled the *plasmodium*. Already the literature of the subject has reached large dimensions, and only the briefest summary of the innumerable researches can be attempted in the present article. The reader will find a most valuable *résumé* of the entire subject, from the pen of D. C. Rees, in the 'Encyclopædia Medica,' vol. vii.

The demonstration by Ross of the part played by the mosquito in the evolution of the malarial parasite affords one of the most interesting chapters in the history of medical progress.

The life-cycle of the parasite (*hæmamoeba* or *plasmodium vivax*) which causes benign tertian fever in man may be taken as a type of the malarial process.

It is found as a small unicellular amœboid mass of protoplasm, in which, after staining, a nucleus and nucleolus may be demonstrated. At first apparently on the surface, and at a later stage within the substance of the red blood-disc, it exhibits amœboid motion, which gradually becomes more sluggish as it increases in size.

When a drop of blood from the finger of a patient who has recently passed through the shivering stage is examined, the plasmodium may be seen as a minute body measuring 1 or 2 μ in diameter, devoid of pigment-granules, and lying upon, or occupying but a small area of, the surface of a red blood-corpuscle. If a drop be examined twelve hours later, the parasite will be found so increased in size as to occupy a third or fourth of the red disc, into which it has penetrated, and into whose substance it projects and retracts its amœboid pseudopodia. Pigment or melanin granules begin to appear at this stage. By the expiration of about forty-six hours the invaded and swollen and now pale-coloured corpuscle is

almost entirely occupied by the parasite, which has assimilated its hæmoglobin, excreting it in the form of coarse pigment-granules scattered throughout its protoplasm. The nucleus of the plasmodium breaks up and surrounds the pigment, which now accumulates in a dense central mass. A short time before the approaching rigor occurs the protoplasm of the parasite will be seen in the process of active segmentation, by which, in a rosette fashion, it divides into from fifteen to twenty or more spores, still encapsuled in the remnants of the red blood-cell, in which stage it is known as a sporocyte. During the rigor these spores are set free into the blood-plasma, when they are called amœbulæ, and, each seizing upon a red blood-disc, the cycle, as just described, begins again.

It will be observed that this mode of reproduction is entirely *asexual*, but side by side with it, in the human body, there is a true *sexual* method of propagation provided for, which, however, is only completed after the parasite enters the mosquito. The insect thus acts as the definitive host to the plasmodium, man being its intermediate host.

The provision for the final sexual method of propagation is not yet demonstrated in completeness in the human blood; but when the red discs are closely examined between the shivering attacks, some are found to contain the plasmodium in its fully matured, pigmented, and spheroidal state, occupying the greater portion of the corpuscle, just as if it were ready to sporulate. Some of these spheroidal masses, however, never show segmentation or sporulation; they are of two varieties—hyaline and granular. The hyaline spheres have flagellating processes which represent the male elements: they are termed micro-gametocytes; the granular spheres, being considered as representing the female elements, are termed macro-gametocytes. In freshly drawn blood the long flagella of the male spheres are thrown out in lashing vibratory movements which last for a considerable period.

These spheroids or gametocytes or gametes, seen in the apyrexial period of benign tertian and quartan fevers, correspond with the crescent bodies found in the blood of patients suffering from the malignant or æstivo-autumnal fever. In this type the gametocytes are found as semilunar bodies, occupying the convexity of a red blood-disc. The crescent of the length of 8μ consists of colourless protoplasm, having in its centre a mass of needle-shaped pigment-granules arranged around its nucleus; in its concavity lie the remains of the almost colourless red cell, which is also stretched over the convex arch or back of the semilunar or crescent-shaped gametocyte. These crescents of malignant fever, like the spheres of the mild tertian and quartan types, are male and female after their kind, and the sexes can be differentiated

by the Romanowsky method of staining. In freshly drawn blood the male flagellating crescents are easily recognised by their lashing movements; and by means of the flagella which break off from them and enter the female, impregnation or fertilisation is effected.

It is held that the spheres in tertian and the crescent bodies in the malignant type of malaria never go through the process of flagellation in the human body, and hence fecundation of the macro-gametocytes by the flagella does not occur till the blood is drawn from the vessels.

It is at this interesting point that the part played by the mosquito becomes of such importance, as demonstrated by the patient and brilliant researches of Ross. The blood of a malarious patient bitten by a mosquito finds its way into the so-called stomach or middle intestine of the insect, where, in a few minutes, as upon the microscopic slide, the gametocytes are set free, a flagellum of the hyaline male sphere breaking off and penetrating a female sphere. The impregnated granular sphere rapidly changes its shape, becomes elongated and sharp-pointed at one extremity like a nail, and exhibits remarkable powers of penetration and motility, in which stage it is called a travelling vermicle or zygote. This penetrates the stomach-wall, rapidly enlarges in size, becoming spherical; in its interior numerous secondary spheres and spherules develop, which ultimately disappear, leaving the impregnated vermicle as a large capsule, which may measure 60μ in diameter, distended with sickle-shaped bodies and projecting as a small bag into the body-cavity of the insect within which it finally bursts. The showers of sickle-shaped sporozoites or filiform young parasites crowd the salivary glands of the mosquito, especially the middle lobe, to which they are carried in the blood-stream. Upon the malaria-laden insect biting a healthy human being, the sporozoites are inoculated along with some saliva, which the mosquito injects for the purpose of preventing the coagulation of its victim's blood. The sporozoite, having thus gained an entrance into the human blood, rapidly develops into the plasmodium as it attaches itself to the red blood-cell, where it undergoes the process of asexual proliferation during several generations, each segmentation or sporulation stage corresponding with a rigor.

The process is essentially the same in quartan, tertian, and pernicious malaria, save that in this latter the gametocytes are of the semilunar or crescent shape as long as they remain sterile in the human blood; but when the blood is drawn by the mosquito, the crescents rapidly become spherical, like the gametocytes of the tertian and quartan parasites before flagellation or impregnation occurs in the insect's body. It is therefore obvious that since the sexual forms—the

gametocytes—remain sterile as long as they are contained in the blood-vessels of the human being, it requires the sexual phase of development in the interior of the mosquito to perfect the cycle of the parasite's existence. Consequently man is said to be the intermediate, and the mosquito the definitive, host of the plasmodium. The period required for the complete sexual development of the parasite in the body of the mosquito is about six to ten days.

As regards the microscopic characters of the *hæmamaeba* or *plasmodium malariae* which produces ordinary quartan fever, it differs but slightly from the *H.* or *plasmodium vivax* of tertian fever; but it has a better defined outline, increases more slowly in size in the red cell, which it never distends and which does not lose its red colour; its pigment is coarser, it has less motility, and finally, in the rosette or sunflower stage, it breaks up, after seventy-two hours, into from six to twelve spores or segments instead of into fifteen or twenty-three, as in *P. vivax*.

Plasmodium præcox or *hæmomenas præcox*—the parasite which causes the pernicious or æstivo-autumnal fever—is much smaller; in its full asexual development-stage it does not measure half the size of a red blood-disc; it has little pigment, and the red cells which it invades often become crenate and shrink in size. The fully matured parasite is usually only found in the blood of the spleen and bone-marrow, and, as already described, the sexually differentiated forms—the gametocytes—are of crescent shape; whilst in the tertian and quartan fevers these appear in the form of large spheres either hyaline or granular.

The mosquito itself requires a brief reference, owing to the important rôle which it plays in malaria. At least twelve genera are described, but only one (*Anopheles*) acts as the definitive host to the three species of malarial parasites which attack man. The genus *culex*—the house-mosquito which may harbour the *filaria nocturna*—closely resembles *Anopheles*, but it never transmits malaria. The mosquito lays its eggs in water or on the leaves and stalks of aquatic plants or floating objects, the ova, larvæ, and pupæ having an aquatic existence; only the mature winged insect enjoys a brief aërial life of four or six weeks' duration. The insect is nocturnal in habit, biting only in the night-time; though Rees points out that some varieties bite in the daytime, and all may do so in dark shady forests. Only the female sucks blood. *Anopheles* is distinguished from its domestic and harmless relation—*culex*—by its preference for rural districts where deeper water is obtainable, and by its attitude when at rest, in which the body is usually at a considerable angle to the plane upon which its legs are placed, the proboscis, thorax, and abdomen being held in a straight line. Of *Anopheles* over fifty distinct species

have been described, nearly all of which have dappled or spotted wings, and it is probable that all the species are capable of acting as carriers of the malarial parasites when an infected human malarial subject is brought within their reach; their ordinary nourishment is vegetable-juices. Manson, speaking of intensely malarious regions which are practically uninhabitable, suggests that some other vertebrate animal besides man may possibly act as the intermediate host of the parasite, or that the parasite may be transmitted direct from one generation of mosquitoes to another without any intermediate host, as is seen in the Texas cattle-tic and the disease affecting the caterpillar of the silkworm.

As already stated, the insect usually remains close to its breeding-grounds, its locomotive powers not being equal to prolonged flights. The mosquitoes can be easily destroyed when in their aquatic stages, and this, with other points in association with its nocturnal habits and the nature of its breeding-grounds, become questions of enormous importance in the prophylaxis of malaria.

TYPES OF MALARIAL FEVER

The fevers produced by the malarial parasites may be put into two classes—viz. First, those which are characterised by *regular* periodical attacks of fever, separated by regular intervals in which the pyrexia is absent. This group includes the types known as *regular intermittent* fevers, which in their typical features are seen in tertian and quartan malaria, or, as they are sometimes called in England, *ague*. The second class contains the *irregular* forms of fever known also as the *Irregular Intermittent*, the *Remittent*, the *Continuous*, and the *Pernicious malarial fevers*. Osler makes a third division, under the heading of the 'Malarial Cachexia,' which he regards as a masked form of the disease in some cases, since it may occasionally be witnessed in patients who have never exhibited the usual symptoms of malarial pyrexia.

TERTIAN FEVER.—This benign form of malarial fever is the most common of all the manifestations of malaria in temperate or sub-tropical climates. It is caused by the invasion of the red blood-cells by the *plasmodium vivax*, whose asexual propagation in the human blood has been described upon page 946.

Symptoms.—After a period of incubation, varying from about six to twelve days or longer, the attack is ushered in with more or less abruptness by a severe rigor. In many cases there is a prodromal period of variable duration, in which some feeling of malaise may be experienced; but in others the symptoms suddenly appear whilst the patient is feeling quite well. The invasion proper or febrile paroxysm is definitely marked by three prominent symptoms—viz. a rigor, fever, and profuse sweating.

The rigor is usually described as *the cold stage*. The patient becomes restless; he may have headache or pain in his muscles and joints, followed by a sensation of cold water being poured down his back. His teeth begin to chatter in the general shivering which shakes his entire body; the movements are transmitted to the bed-clothing, and even the bed and immediately surrounding objects may be set into vibration. His face becomes pinched and cyanosed, respiration and phonation are interfered with, and the surface of the body becomes icy cold. The pulse is small, hard, and quickened; intense headache, severe muscular pains, and often smart vomiting, are present. At this period, though the patient feels benumbed with intense cold and covers himself with all the available clothing within his reach, the thermometer may in the rectum or axilla register as high as 105° , and even immediately before the supervention of the rigor the temperature, if investigated, will always be found to be somewhat elevated. The duration of the cold stage is variable; it may last from a quarter of an hour to an hour or more.

The hot stage is ushered in with a sensation of flashes of heat, which rapidly passes into a burning fever in which the skin feels hot to the touch and very dry. Blueness disappears and gives way to redness or a dusky yellow tint; the face is congested, the eyes suffused; the mouth and tongue are parched, and thirst is complained of. The general restlessness increases, and is often aggravated by bilious vomiting, which may have set in during the cold stage; diarrhoea is not unusual. The pulse is full and bounding, the headache and pains in the limbs continue, and in severe cases there may be active delirium. The axillary or rectal temperature may not register above that noted at the termination of the cold stage, but usually it reaches its maximum within a couple of hours after, when 106° or 107° may be noticed; more frequently, however, it does not exceed 104° or 105° . At this period the spleen, and sometimes the liver, will be found somewhat enlarged. The duration of the hot stage is variable: it commonly lasts three to four or five hours, after which

The sweating stage sets in, with beads of perspiration appearing about the face and head, soon extending to the entire surface, which becomes bathed in a profuse sweat, saturating the bed-clothes and reducing the temperature rapidly to the normal. All the symptoms speedily give way, and by the end of a couple of hours the patient usually falls into a sound and refreshing sleep, from which he awakes in ten or twelve hours after the initial rigor feeling comparatively well, only some herpetic eruption about his lips or nose remaining.

The intermission in this variety of malarial fever lasts for about thirty-six to forty hours, during which the temperature

is usually sub-normal, and the patient feels quite well and able to pursue his ordinary avocation. Upon the third morning—that is, in about forty-eight hours after the commencement of the first paroxysm—he is seized with a rigor and passes through the same stages of shivering, fever, and sweating exactly as before. If untreated by quinine, the paroxysms may continue to appear in obedience to the same law of periodicity for an almost indefinite time, till the patient is reduced to the condition of typical malarial cachexia, with its enlarged spleen, anæmia, and tendency to fatal hæmorrhages. As a rule, however, even when benign tertian malaria remains untreated, the natural course of the disease seems to exhibit the phenomenon known as *postponement*, in which the paroxysms are divided by gradually lengthening apyrexial periods, and, becoming less severe in degree, they may entirely cease, unless a new exposure to infection occur.

When quinine is administered a few hours before an expected seizure, though the parasites are destroyed the paroxysm is not prevented; but the new generation of young spores do not develop, and a second paroxysm may never occur, the patient rapidly convalescing.

Double Tertian Fever.—As just described, the parasite which produces ordinary benign tertian fever runs its course of asexual development in the human blood inside a period of forty-eight hours, by which time it has arrived at the stage of sporulation or segmentation, which phase corresponds with the hour of the advent of the rigor. When two generations of the same parasite are present, owing to a *double infection*, if they sporulate on alternate days the type of fever is then known as a *double tertian* or *quotidian*, since a paroxysm will appear daily. This is a very common type of fever in temperate climates. The double infection is not to be confounded with *mixed* infection, in which two different species of parasite may at the same time infect the blood of the same patient. Moreover, as will be mentioned later, a quotidian type of fever may be produced by three different generations of the quartan parasites sporulating daily. Thus quotidian fever may be the result of double infections of the tertian parasite or triple infections of the quartan.

QUARTAN FEVER.—This is a regular, intermittent form of malarial fever like the benign tertian, but caused by a different plasmodium (*hæmamoeba malaricæ*) which requires a period of seventy-two hours for the complete cycle of its asexual development in the human blood. It is not so common as the tertian, and it is, upon the whole, milder or less liable to be followed by serious results; but it does not respond quite so readily to quinine-treatment, and, as the parasite can remain latent for indefinite periods in the blood, paroxysms have been frequently observed long after the patient has left

the malarious district, though its average period of incubation is a little under a fortnight.

Symptoms.—These may be regarded in every way as identical with those seen in the cold, hot, and sweating stages of tertian, with the important difference that in a typical infection the rigor sets in upon the fourth morning after the rigor of the initial paroxysm, in cases where there has been a *single* infection with one generation of parasites.

Double quartan fever shows an interesting type of pyrexia: two generations of parasites, each sporulating after seventy-two hours, produce paroxysms upon two successive days, the third day being apyrexial.

Treble quartan, with its three separate generations, is characterised by a paroxysm every morning; in other words, it is a quotidian fever, clinically identical with the quotidian type produced by a double infection of tertian parasites.

Mixed infection by quartan and tertian parasites occurs, as does also a mixed infection with the quartan and the parasite of malignant malaria. In rare cases, all three species of parasite may infect the blood at the same time, causing very confusing types of intermittency, remittency, and continuity in the pyrexia.

TROPICAL, MALIGNANT, OR ÆSTIVO-AUTUMNAL MALARIA.—The fevers produced by the malignant parasite (*hæmomenas præcox*) are designated under the above headings; they are known also under several names indicative of the peculiarities in the type of the pyrexia, as *Remittent*, *Irregular*, or *Continued Malarial Fever*, and under such names as *Pernicious*, *Bilious*, and *Typhoidal Remittent*, and *Adynamic Fever*, as descriptive of certain other marked clinical features.

As the terms 'intermittent,' 'remittent,' and 'continued' occur repeatedly in the description of this group of fevers, it may not be out of place to remind the reader what is implied exactly by the use of each term. In the *intermittent* type of fever, the temperature between the periodical attacks of pyrexia falls to the normal or below it, and remains so till the next paroxysm. When the temperature is constantly above the normal, but the drops or regular falls in its variations are so marked as always to exceed a degree and a-half F., the type is styled *remittent*; whilst in the *continued* or *continuous* type the temperature, though constantly above the normal, drops usually in the morning to an extent never greater than from a-half to one and a-half degree below the evening rise.

Malignant malarial fever is most common in the tropics, where it may flourish all the year round. When it occurs in temperate regions, as in the Roman Campagna, it makes its appearance in the late summer and autumn; hence its Italian name—*æstivo-autumnal*. It is broadly separated from the

tertian and quartan types, which show such remarkable periodicity, by its frequent tendency to irregularity in type of pyrexia as implied in its numerous synonyms, intermittency very frequently giving place to irregular remittency, or a more or less regular continuous type of fever. With these differences in type are associated a gravity in the symptoms, and a tendency towards a fatal result or the establishment of a high degree of cachexia, notwithstanding, often, the early administration of the alkaloid which exerts such a specific action over the other parasites.

The diversity of the symptoms is suggestive of infection by different parasites, and Mannaberg maintains that there are three distinct species of parasites which produce the malignant type of malaria. One thing is certain—whether the parasites are mere varieties or distinct species, they all have their gametocytes of the shape of the crescent or semilunar body. The parasite has been already described upon page 949. Its period of asexual development is usually about forty-eight hours, but the variations in this respect are very marked, whether owing to the parasites being of different species or the infections being mixed or doubled. The life-history is not so easily studied, because the plasmodium, before its segmentation-stage, is rarely found in the blood removed from the finger, having retired into the blood in the deep viscera, chiefly in the spleen, where it can be obtained in large numbers. In the early stages, soon after the advent of the paroxysm, they are found in the peripheral blood, and several may be seen as small active amœbæ, seizing upon the same red blood-cell—a phenomenon which is very seldom witnessed in the benign intermittent fevers. Each fully developed plasmodium divides into from five to twelve minute spores, and often only occupies a small portion of the red disc in an eccentric manner. The crescent bodies are not met with in the blood from the finger till after the expiration of about a week, and, as in the tertian and quartan fevers, they never flagellate inside the human blood-vessels.

Symptoms.—After a period of incubation much shorter than that of the benign intermittents—*i.e.* in about three days, on the average—the patient complains of lassitude, general pains, and headache, after which there may be a distinct rigor, with abrupt rise of temperature; more frequently, however, there is no very well-marked cold stage, the fever rising gradually and falling gradually afterwards during the profuse sweating stage.

The intermediate or hot stage is very prolonged, often extending over twelve hours. The entire paroxysm (cold, hot, and sweating stages) lasts from twenty to thirty hours—*i.e.* from two to three times the duration of the paroxysm of the benign intermittents. The apyrexial period or inter-

mission is very variable ; it is always shorter than in tertian attacks, and in some cases the fever may be of the remittent or continuous type throughout. The most common variety of the disease recurs after a true intermission of variable duration at the end of twenty-four or thirty-six hours from the onset ; as successive paroxysms occur the intermission becomes shorter and shorter, till the temperature ceases to fall to the normal, the intermittency then passing into remittency as the phenomenon of anticipation becomes more complete. As the disease progresses in favourable cases the remittent type of temperature may give place to an intermittent type.

In those cases styled 'remittent' from the onset the temperature does not fall to the normal, but remains high, with drops of a degree and a-half or more (F.).

In those cases of malignant fever where there is a more or less complete daily intermission, the temperature may show the quotidian type almost identical with the quotidian fever produced by double tertian or triple quartan infection.

As a rule, the temperature does not reach the height registered in the sharp cold stage of the benign intermittents, though it is always more prolonged ; but in the pernicious cases it not very uncommonly registers 110° . The symptoms during the attacks vary considerably in degree, though in the main they resemble those seen in tertian fever, with the remarkable difference emphasised by Rees that the subject of the malignant plasmodium is liable at any moment to a pernicious attack. Where the temperature tends to follow the continuous type the symptoms are obviously altered by the prolonged fever, and soon the patient begins to look severely ill, presenting the usual appearance of a patient suffering from typhoid fever, with dry brown tongue, some delirium, and marked enlargement of the spleen. More usually, however, the variations in temperature exceed those seen in the typical typhoid-chart, so that the type becomes that of remittent fever. Such cases are described as *Typhoidal remittent* and *Bilious remittent* fevers, the former being but a more severe form of the latter. There is usually much bilious vomiting, sometimes accompanied by diarrhoea and generally jaundice or an icteroid tinge of the skin and conjunctiva, and a higher range of temperature than that met with in the simple malignant cases, though lower than the pyrexia of the pernicious attacks. In very feeble and debilitated individuals an *Adynamic* type is met with, especially in the subjects of chronic alcoholism.

After a variable period sometimes extending over more than a couple of weeks, if the patient escapes a pernicious attack or does not succumb in the typhoidal remittent form of the disease, his temperature may gradually or even rapidly

fall, and a speedy convalescence may set in ; but usually the recovery is much slower than after the benign forms, and his temperature may long remain sub-normal.

(For the relations of *Blackwater Fever* with malaria see page 106.)

PERNICIOUS MALARIAL FEVER.—This should not be regarded so much as a distinct type of fever as a modification of the malarial process which is liable to supervene at any stage of malignant fever. Though a pernicious attack is almost always associated with the presence of *plasmodium* or *hæmomenas præcox*, nevertheless it has been known to occur in the tertian and quartan fevers, though very rarely. These attacks are usually only met with in very hot climates, but they are not uncommon in the autumn in the worst malarious districts in Italy ; and occasionally they are witnessed during the relapses which occur in England amongst patients who have recently suffered from malignant fever in the tropics.

The theory of their causation which is generally accepted is that the attacks are produced by the sporulation of the parasite in the blood-vessels of the viscera or brain. Many different forms of pernicious attack are described.

In the *Hyperpyrexial* variety of attack, the patient, during a paroxysm, or more usually soon after this has passed off, is seized with smart delirium, which passes rapidly into coma, soon followed by a fatal issue, the temperature registering 110° or more. In some examples described as *comatose* attacks the initial symptom may be a brief delirium, rapidly passing into coma, with a high but not hyperpyrexial temperature, from which the patient may recover after a period of profound unconsciousness lasting for twelve or twenty-four hours. He may, however, die in the coma, or, after recovering, be again seized with another attack, which may prove fatal with or without hyperpyrexia.

These forms of pernicious attack have been found associated with gross lesions caused by the parasite in the blood-vessels and surrounding brain-tissue. Sometimes the cerebral lesions cause paralysis, aphasia, amaurosis, or mania, producing a clinical type designated after the most prominent group of symptoms.

Where the parasites gravitate towards the gastric or intestinal vessels, the so-called *Algide* group of pernicious attacks result. These are the *Gastric*, *Cholericiform*, and *Dysenteric* varieties, in which, owing to the persistent vomiting and watery or dysenteric diarrhœa, the symptoms of gastritis, cholera, and dysentery are simulated. A *syncopal* variety, in which the heart's action becomes alarmingly weak or stops entirely, has been described.

Any of these algide pernicious attacks may supervene at any stage of the disease, and they may, especially in old

people, be entirely *apyrexial*; but often they pass into the asthenic or adynamic type before death results.

Pathological Anatomy of Malaria.—The condition of the blood has been already described; large numbers of the red cells are destroyed, and there is therefore marked hæmoglobinuria, greater than the mere number of ruined cells will account for. Black and yellow pigment is showered into the blood at the sporulation-period, and accumulates in the liver, spleen, marrow, and brain. The pigment-granules are constantly also found in the leucocytes. In chronic cases there is extensive enlargement of the spleen, which is firm from great thickening of its capsule and trabeculæ, constituting the so-called 'ague-cake.' In recent cases the organ is softened and of a raspberry-juice colour, and enormous numbers of parasites are to be found in its blood. The liver is enlarged, and a large amount of pigment may be found in it, but few parasites are visible, whilst the intestinal blood-vessels may be loaded with them. Since albuminuria is often present during the attacks, it is not surprising to find sometimes evidence of nephritis after death in both the recent and chronic types of malaria.

MALARIAL CACHEXIA

This is a condition which supervenes after repeated attacks of any of the malarial parasites, but chiefly after the malignant one. Rarely does it follow the paroxysms caused by a single exposure, unless treatment has been neglected and the patient has been in a debilitated or otherwise diseased condition before his fever. In the *abortive* or *larval* attacks, which are believed to occur with frequency in all malarious districts, there may be no evidence of chills, fever, or sweating, the only clinical manifestations of the invasion of the parasite being furnished by the obvious cachexia; this is most frequently observed in children. The condition is characterised by a secondary anæmia, with a faint icteroid tinge of the conjunctiva and a sallow parchment-like skin, more or less wasting being always present. The usual accompaniments of severe anæmia are well marked—viz. breathlessness, palpitation with cardiac murmurs, œdema of the legs, and a tendency to hæmorrhages from the mucous surfaces and into or under the skin and retina. There may be a slight degree of fever, especially if the paroxysms have been recent, in which case the parasites will be found in the blood. The most important evidence is found in the spleen, which is constantly enlarged, often to an enormous extent, and in old cases it is hard, with a well-defined margin. From splenic leucæmia it is easily differentiated by the great oligocythæmia and absence of leucocytosis, the polymorphonuclear leucocytes being often diminished.

The condition may remain for a long time, and finally disappear on the removal of the patient to a healthy district, though the spleen seldom, if ever, regains its normal dimensions. It is, moreover, liable to rupture and cause death from internal hæmorrhage; and in children a fatal result is not rare from marasmus and exhausting diarrhœa. In adults a condition of invalidism in which exhausting neuralgias are a prominent feature may become very chronic or permanent.

COMPLICATIONS OF MALARIA

The complications of malaria are numerous. Dysenteric diarrhœa is common, probably from the presence of the parasites of both diseases at the same time, since dysentery is an affection commonly met with in the zone of malaria. Typhoid fever is also frequently coincident; hence the term 'typho-malaria.' It would appear that any disease occurring in a patient who has once suffered from severe malaria may exhibit the symptoms of his new affection in a form modified by the old malarial condition. Thus, in a patient under the writer's observation for many years the passage of a urethral sound for an old stricture was invariably followed by a most alarming rigor of about an hour's duration, with a prolonged hot stage and drenching perspirations, which he always maintained was a recurrence of his original malaria of many years before. The entire paroxysm occupied ten or more hours, and was certainly unlike any urinary rigor met with. This was before the modern methods were employed for the demonstration of the parasites in the blood; he never exhibited any other symptoms of malaria. A similar phenomenon is witnessed in children who have suffered from pertussis, any bronchial irritation long afterwards producing the characteristic spasmodic cough and whoop. This probably affords a clue to the remarkable periodicity which characterises some forms of neuralgia which may attack old malarious subjects.

Pneumonia and orchitis are not rare complications, and the forms of paralysis mentioned as occurring in one variety of the pernicious attacks may appear in benign malaria, and sometimes may remain.

¶ *Relapses* are common, and it should always be borne in mind that these may occur in patients who have years previously left the tropics to reside in a non-malarious climate, the parasite in such cases remaining latent or dormant for long periods in some internal organ.

Diagnosis.—The recognition of the parasite in a drop of fresh blood under a $\frac{1}{12}$ -inch oil-immersion lens and a sub-stage condenser, is comparatively easy in the hands of those accustomed to blood-examinations. The various staining

methods may be employed in doubtful cases. The only difficulty in the diagnosis arises in the malignant type of the disease if the examination is made at the period when the parasites are very few in the peripheral blood—in which cases they will be detected swarming in great numbers in the blood from a splenic puncture.

Even when the malarial parasite is not found in a given specimen of peripheral blood in any form of malaria, the presence of pigmented leucocytes or a relative increase in the large mononuclears is very suggestive. In benign tertian and quartan malaria there is usually no difficulty in the diagnosis, the periodicity of the attacks and the feeling of complete relief during the intermission being so marked.

Typhoid fever may so simulate malignant remittent malaria that a diagnosis is only possible after a very careful blood-examination. When both diseases occur together, the malarial parasite may be demonstrated by the microscope, and the presence of typhoid fever be shown by Widal's test.

Ulcerative endocarditis is met with in a form in which the symptoms are practically identical with intermittent malaria. In one case seen by the writer, and occurring in a patient recently resident in a malarious district, the diagnosis was suddenly confirmed by the appearance of an aortic regurgitant murmur and the plugging of a large artery.

Pyæmia and *liver-abscess* may be differentiated by the irregularity of their periodicity, by the local signs of the abscess, and the blood-condition, which always shows marked leucocytosis. It is well to remember that the septic conditions which simulate malaria are nearly always associated with a marked leucocytosis, which is rarely observed in any form of malarial fever.

Kala-azar is now differentiated conclusively by the presence of the Leishman-Donovan bodies in the blood from a splenic puncture.

Quinine is a valuable test in all doubtful cases, since every form of malaria will yield to its judicious administration. Often the first doubt as to the nature of a non-malarious fever occurring in a malarious district is aroused in the physician's mind by the failure of the drug in modifying or removing the pyrexia.

It should be remembered, however, that large doses of quinine may cause amaurosis, which may be mistaken for malarial amblyopia.

Prognosis.—In the benign intermittents recovery may be confidently expected when treatment has been resorted to early; and this may be said also of the æstivo-autumnal fevers of temperate climates, with, however, some reservation. The malignant fevers of the tropics, when pernicious attacks occur, are very generally fatal.

Treatment.—The prophylaxis of malaria, owing to the brilliant discoveries of Laveran, Ross, Celli, Casagrandi, Koch, Manson, and many other workers in this important field of research, has become a practical problem. The points in connection with the destruction of the *Anopheles*, which is the only known definitive host of the malarial parasites, have been incidentally referred to in the remarks on the drainage of malarial districts. Successful attempts have been made at the destruction of the mosquito whilst in the aquatic stage of its existence. This has been easily accomplished by covering the surface of the pools in which the larvæ have been hatched by a film of petroleum-oil or any tarry solution, or by the addition of very small amounts of coal-tar products, or even of sea-water.

Since *Anopheles* is largely nocturnal in its habits in bright weather, decided results have been achieved in the most intensely malarious districts by the exclusion of the insects from the sleeping compartments by means of mosquito-netting.

The value of quinine as a prophylactic has been long known, and European residents in malarious localities have protected themselves for long periods by a daily amount of not less than 5 grs.—preferably 8 to 10 grs.—administered in divided doses spread as evenly as possible over the twenty-four hours. Arsenic is also said to be prophylactic. Of the different methods of employing quinine as a prophylactic, that known as Celli's consists in the administration daily of two tabloids of the bisulphate or acid hydrochloride, containing 3 grs. each. Plehn's method, known as that of 'double prophylaxis,' consists in the administration of 8 grs. every fourth and fifth, or every fifth and sixth, day. Koch's plan, known as the 'long interval prophylaxis,' consists in giving 15 to 24 grs. upon two consecutive days at intervals of eight to ten days.

It is hardly necessary to say that the spread of the disease is much influenced by the rigid isolation of all infected human beings in buildings made proof against the entrance of the mosquito by netting and other means.

In temperate countries the prevalence of malaria is only maintained by the relapses which occur in the warm season.

As regards the treatment of the malarious patient, quinine is the only drug to be relied upon once the parasites have gained admission to the blood. The young brood when showered free into the blood-plasma are much more susceptible to its lethal influence than are the intracorpuseular forms, and a single large dose (15 grs.) introduced into the circulation just before the paroxysm of the benign intermittent fevers will effect the destruction of the great majority of the young parasites. Though the rigor will follow in due course, it may not be succeeded by another; but it is always

advisable to continue the drug for at least a month. The malignant parasite is more resistant, and over its intracorpuscular forms quinine possesses much less power. In the treatment of the malaria produced by this parasite it is absolutely necessary to saturate the blood as rapidly as possible with the drug; hence hypodermic or intravenous injections of solutions of the very soluble acid hydrochloride should be resorted to without delay: 5 or 10 grs., dissolved in 20 mins. of sterilised water, may be injected every four or six hours. The administration of the less soluble sulphate, especially in pills and tabloids, should be abandoned in all severe cases, though its prophylactic value is probably greater. Even in the benign intermittents, though some authorities recommend the postponement of quinine till the paroxysm has shown itself, it is usually wise to administer the drug by the mouth, as soon as the patient comes under observation, in one full dose of about 15 grs., followed by 5 grs. every four or six hours. When the hypodermic method is employed in malignant cases, the needle should be deeply inserted into the gluteal muscles. A large dose in solution may also be administered into the rectum, but rarely is it necessary, even in malignant cases, for the daily dose to exceed 45 grs. Quinine seems to have no influence over the gametocytes; and since these bodies appear only to be produced after the asexual forms have reproduced themselves in the blood, it is probable that early resort to the administration of quinine lessens the chance of their formation and tends to check the spread of the disease by the mosquito. The liability to relapses is diminished in proportion to the promptness and thoroughness of the saturation of the blood by quinine, and to the length of the period during which its administration is continued afterwards. Methylene-blue has been successfully employed as a substitute for quinine in cases where the latter drug cannot be tolerated.

The phases of the paroxysm are to be treated upon general lines; during the rigor warm blankets and warm draughts, and in the hot stage cold or tepid spongings, may be resorted to. The advent of coma or hyperpyrexia in a pernicious attack should be met by the remedies indicated in sunstroke, which latter condition it may closely simulate.

The treatment of the malarial cachexia is little influenced sometimes by quinine; but this drug should always be combined with arsenic—the only agent which seems to exercise a marked effect upon the dyscrasia. Iron is also valuable when from time to time it is included in the combination. Removal from the malarious district to a temperate climate is essential.

MALIGNANT PUSTULE (ANTHRAX)

This is a lesion produced by the admission of the bacilli or spores of *anthrax* into scratches or abrasions in the skin.

Bacteriology.—The *bacillus anthracis*, which produces the *Splenic Fever* or *Anthrax* of animals, is one of the most widely distributed and malignant of micro-organisms. It is a motionless rod-shaped body, measuring from 5 to 25 μ , but usually it is seen of the size of about the diameter of a red blood-disc; in the blood it increases with great rapidity. Its virulence is easily destroyed or attenuated, and in this condition its injection into the blood confers immunity to the disease in susceptible animals for considerable periods. When in the blood it is usually unable to sporulate, but outside the body, in presence of free oxygen, spores are produced which possess extraordinary powers of resistance to great heat and antiseptics. These may retain their virulence for many years, speedily developing into bacilli when introduced into the blood by the bites of insects, or through the alimentary canal in the fodder of cattle or more rarely in the food of man, or when inhaled as dust from the hairs and wool of affected animals. In cattle the disease is most fatal, and, owing to the vitality of the spores, epidemics are stamped out only with the greatest difficulty. Splenic fever is met with in herbivora and in every class of vertebrates, and in all regions of the earth. In England the prevalence of anthrax in man in the West Riding of Yorkshire and districts like Northampton and Leicester is explained by the nature of the industries in these localities. It is endemic in cattle in South America, Russia, Western Asia, and Persia.

Etiology.—In man anthrax is conveyed by the spores or bacilli from living diseased cattle, as is witnessed by the occupation of its victims—stablemen, drovers, farmers, shepherds, veterinary surgeons, and cattle-dealers. Dead animals as sources of infection convey the disease to slaughter-house-men and butchers; in a few cases the infection has been transmitted in wounds received by the surgeon in a *post-mortem* on an anthrax-subject.

In this country anthrax is most commonly conveyed to those whose occupation brings them into direct contact with the hides, wool, hair, and other imported products obtained from diseased animals; hence the disease is usually met with in tanners, furriers, wool and hair sorters, plasterers, mattress and brush makers, and sometimes in rag-pickers.

Three well-marked types of anthrax are described whose symptoms depend upon the route by which the bacillus or its spores gain admission to the human body. These are Cutaneous Anthrax, Intestinal Anthrax, and Wool-sorters' Disease or Pulmonary Anthrax.

CUTANEOUS ANTHRAX

This is known as *Malignant Pustule*, and in France as 'Charbon,' and, as already stated, it is the result of infection by the anthrax spores or bacilli becoming implanted in the sound skin by the bite of an infected insect, or by inoculation, or more usually by the organism being brought into contact with an abraded or wounded surface. Blood from a case of splenic fever, on exposure to air, moisture, and heat, rapidly produces spores, which probably play a more important rôle in infection in all forms of anthrax than do the more easily destructible bacilli.

The lesion may in some cases remain entirely local; more frequently, however, there is evidence of general infection—i.e. *Anthraxæmia* is also present.

Symptoms.—The inoculation nearly always occurs upon an exposed part, as the face, neck, or hands, and rarely is there ever more than one focus. The period of incubation is sometimes only a few hours, after which a red, itching, and tender papule appears; this at the expiration of twenty-four hours has become a vesicle full of blood-stained serum, around the base of which redness and much brawny swelling appear. The bursting of the vesicle upon the second or third day is followed by the production of a dark-brownish dry eschar, which is somewhat depressed and surrounded by an elevated ring of small secondary vesicles. The inflammatory œdema or brawny swelling increases and becomes very extensive, especially if the region about the eye or the neck is the site of inoculation; sometimes it spreads over the entire head and neck in a few days, preventing both breathing and swallowing by the general infiltration of the lymphatics and soft parts.

Pain is usually very slight or entirely absent, and suppuration does not occur till after the separation of the eschar at a late period, if the patient survives. There is often remarkable absence of any feeling of serious illness or anxiety—a condition which is constantly to be met with in all forms of anthrax.

In a considerable percentage of cases, fever and constitutional disturbance afford evidence that the blood has become invaded; but Bell emphasises the fact that the general symptoms bear no relation to the amount of local disease. The fever, which usually does not appear for some days, has no special characters, and may soon subside; but the subsequent symptoms may be indicative of an invasion of the stomach, intestines, brain, lungs, or heart, as seen in the types of internal anthrax. In many cases the bacillus remains confined to its original site in the skin, and general infection does not occur. The vesicles in the early stage contain the microbes in great numbers, but later, as the eschar or slough increases in size, they are found with difficulty.

The severity of the disease varies very greatly, owing to the individual resistance of the patient; in mild cases recovery may set in after a week by the spontaneous shedding of the eschar. Death may occur, however, as early as the third or fourth day; it is rarely protracted till the suppuration-stage, which commences about the end of a fortnight after the separation of the slough, when pyogenic organisms gain an entrance. In some cases described as *malignant anthrax oedema*, no papule, vesicle, or eschar appears, the only signs being those of extensive oedema; this generally begins about the eyelids or neck.

Diagnosis.—The most reliable diagnostic sign is the presence of the vesicle, followed by a dry eschar without pus or any sloughing soft core, which differentiates malignant pustule from *boils* and *carbuncle*. The discovery of the bacillus in the vesicular contents is conclusive, especially if the fluid be inoculated into mice, in which animals death follows in forty-eight hours. An important element in the diagnosis will be the occupation of the patient, when this exposes him to the handling of foreign hides, furs, &c.

Prognosis.—This is most difficult, owing to the fact already mentioned that it is impossible to judge of the gravity of the case by the extent of the local lesion. The outlook is much more serious when the site of inoculation is on the neck or face; nearly half of all the neck-cases die, whilst Hamer's statistics show a mortality of only 12 per cent. when other regions were infected. In figures collected from South Africa and the Argentine, the mortality appears to be under 1 per cent. The probable explanation of this remarkable discrepancy may lie in the degree of immunity conferred by previous inoculations, which have been so slight through attenuation of the bacilli as to be unnoticed; though Bell attributes the slight mortality to the increased light and heat, and to the activity of the antagonistic septic organisms in these countries.

Treatment.—The site of the local inoculation should be immediately excised either by the knife or cautery, cutting wide of the diseased tissue. Perhaps the most efficient method of excision, where this is possible, would be the removal by the knife, followed by the searing of the wound with the cautery. In some cases excellent results seem to have followed injections of a solution of carbolic acid (1 in 15) into the skin around the vesicles. In all cases the wound or eschar should be dressed with sublimate lotion. Antiseptic solutions, in which the spores might remain for indefinite periods without injury, are sufficient immediately to destroy the bacilli. The injection of Sclavo's antitoxic serum, when this is available, should be always tried.

INTESTINAL ANTHRAX, or MYCOSIS INTESTINALIS

This is a very rare form of anthrax fever, setting in with acute symptoms of gastro-enteritis, not unlike some forms of ptomaine poisoning. The infection in these cases reaches the stomach through eating anthrax-flesh, or possibly through drinking the milk from a diseased animal, or swallowing the spores, which have accidentally contaminated food or water. Ordinary cooking will completely destroy the anthrax bacillus, but the spores are uninfluenced by the process, and they may even resist the digestive process in the human stomach. The symptoms may be entirely gastro-intestinal, death occurring in forty-eight hours from exhausting vomiting, purging, intestinal hæmorrhage, and enlargement of the spleen, without fever; but if life be prolonged, pulmonary signs, as cyanosis and dyspnœa, may supervene, or the patient may die comatose. The disease is very likely to prove fatal. When a diagnosis during life is possible, intestinal antiseptics should be administered freely. Muskett records marked success from large doses of ipecacuanha.

**PULMONARY ANTHRAX, WOOL-SORTERS' DISEASE,
or ANTHRACÆMIA**

This is an internal form of anthrax, met with in the operatives in woollen factories and mills where horsehair, mohair, and alpaca are manufactured. Bell points out the rarity of the disease in factories in which the imported fleeces are received in a greasy condition, and its comparative frequency where the dry hair or skins of the goat or other animal fills the atmosphere of the factory with the spores attached to dust or fine hairs. In Austria the disease is comparatively common in the rag-pickers employed in the paper-industry.

The infection occurs through the admission of the bacilli, or more probably of the spores, to the respiratory system.

Symptoms.—These may be almost entirely latent, the patient only showing signs of collapse and cyanosis a few hours before death, whilst protesting that there is little or nothing the matter with him.

Usually there is some chilliness, vomiting, diarrhœa, muscular weakness, and marked frequency and feebleness of the pulse. Though the skin may be cool and moist, the temperature in the axilla may register a couple or more degrees above the normal, and in the rectum, even when the axillary temperature is normal, 105° may be registered. Though the lungs are always invaded, the pulmonary symptoms and signs may be so slight at first as to be overlooked; usually there is a sense of oppression or tightness of the chest, with slight cough and absence of expectoration.

The respirations, however, become frequent; severe pain in the chest is complained of in a small percentage of cases, though the auscultatory signs are only indicative of a mild bronchitis; pneumonia and pleuritis are rare, except in the protracted cases.

Without further development of signs or symptoms of any constitutional disorder save increasing weakness of pulse, dyspnoea, lividity, and coldness of the skin, the patient rapidly passes into a condition of collapse, and may die inside twenty-four hours after the first symptoms of illness. About four-fifths of the fatal cases terminate within one hundred hours of their onset. Delirium, preceded by headache and sometimes followed by convulsions, occurs in a considerable percentage of the cases before death, and even in these cases the rectal temperature is usually only a few degrees above the normal. Consciousness to the last is, however, maintained in the great majority of cases.

Pathological Anatomy.—The lungs are found deeply congested and dark-coloured, extravasations of blood being frequently present, the right suffering more than the left, according to Bell. The heart is softened, the endo- and pericardium showing ecchymoses. The blood is found to be almost black, which gives a livid discoloration to the skin after death. Gelatinous oedema of the cellular tissue is observed in various regions, and serous effusions into the cavities of the body constantly occur. The bacilli are found in the blood and in all the viscera. The spleen is almost always enlarged, but seldom to any great extent; it is softened and friable, and almost black in most cases. The lymphatic glands in the thorax and abdomen are also often similarly affected, and extravasations between the membranes of the brain and cord are commonly observed.

Diagnosis.—From the description of the symptoms it is obvious that diagnosis is often impossible during life. Where the occupation of the patient suggests infection from the anthrax bacillus, the examination of the blood affords the only means of verifying the diagnosis. Even where the microbe cannot be detected in it, a subcutaneous injection into a mouse or guinea-pig will produce a fatal result.

Prognosis.—This is always grave. Bell's experience proves that the outlook is better in the cases beginning with a smart fever, the high temperature being indicative of natural resistance to the bacillus, as is also the supervention of pneumonia and pleurisy. If the patient survive for ten days his chances of recovery are good, since death is comparatively rare after this period.

Treatment.—Prophylaxis is of great importance; the careful disinfection of all fleeces, hair, wool, &c., coming from the foreign countries in which splenic fever is endemic

or epidemic, should be insisted upon. An animal rendered immune by Pasteur's method of injecting attenuated virus in gradually increasing doses soon has its serum charged with antitoxin. This is a certain *preventive*, if injected into animals before the anthrax bacilli have been inoculated. Sclavo's serum has been successfully used in a large number of cases of all varieties of anthrax with considerable benefit, and there are fair reasons for hoping that, by improved methods, serum-therapy will secure for the disease a rational and efficient treatment.

No other agent or drug has any specific effect upon the bacilli in the human blood. Hence the treatment consists in the exhibition of such remedies as are indicated for the relief of the most urgent symptoms; and since cardiac failure is always pronounced, stimulants, combined with strychnine or ammonia, and cardiac tonics, should be freely administered.

MALTA FEVER

This is a non-contagious endemic fever pursuing a chronic irregular course, characterised by neuralgic and joint-pains, profuse perspirations, and marked tendency to relapses, and accompanied by splenic enlargement, anæmia, and constipation.

Distribution.—The disease is usually met with in the islands of Malta and Sicily, and the regions bordering upon the Mediterranean, and also in some parts of China, India, and Rhodesia; recently its presence in an endemic form has been demonstrated by Birt in the Orange River Colony. Its synonyms are as numerous as the localities in which it is found. Thus in Gibraltar it is styled *Rock fever*; in southern Italy it is known as *Neapolitan fever* and *Sicily fever*, the most common name, however, being *Mediterranean fever*; owing to the type of the fever, especially during the relapses, it has more recently been styled *Undulant fever*.

Etiology.—The cause of the fever is the presence of the *Micrococcus melitensis*, a microbe which does not cause fermentation of glucose or produce coagulation in milk, though it has the power of rendering the milk alkaline. It is decolorised by Gram's stain, and agglutinates with a specific serum in dilutions of 1 in 1,000. The microbe has been recently discovered in the milk of goats at Malta. According to some authorities, it is spread by a mosquito which breeds in the pools of salt water on the shores of the Mediterranean.

It is certainly to be found in the blood of the spleen in all severe cases of the disease in man. The disease is prevalent in the hotter months, when it may become epidemic, remaining endemic during the cool season. It affects males and

females equally, but most frequently attacks young or middle-aged healthy subjects.

The Commission presided over by Colonel Bruce has demonstrated the fact that, in the great majority of cases, the disease is spread by the milk of the goat, though there may possibly be under exceptional circumstances transmission of the disease by air, water, or mosquitoes. It has also demonstrated the correctness of the earlier conclusions arrived at by Louis Hughes, who, according to the authority of Colonel Kilpatrick, first isolated the coccus from the goat. The cocci have also been shown, by means of the application of the agglutination-test, to be present in a considerable number of apparently healthy individuals in Malta, and it has been shown that a considerable percentage of these ambulatory cases may be the means of disseminating the disease through the micro-organisms excreted in the urine.

The microbe is found in the milk of about 10 per cent. of the goats in Malta, and it is calculated that about half of the total number of goats in the island are infected; the mules and dogs are also infested by the coccus. The cocci are freely eliminated by the kidney, and appear in the urine of patients from the fifteenth day of the disease till convalescence is completed.

Morbid Anatomy.—The chief feature is the constant enlargement of the spleen, which is usually very considerable, often weighing two or more pounds. It is soft, friable, and intensely congested, and contains the microbes in great numbers, though these are less easily found in the general blood-stream. The mesenteric glands and Peyer's patches are slightly, if at all, changed, showing a marked contrast to the condition found in typhoid fever. The liver and kidneys also generally show marked engorgement, and the cocci are found in both these organs as well as in the lymphatic glands.

Symptoms.—The *incubation* period is about five to ten days, as seen in the cases of new arrivals; it may under certain circumstances be prolonged to four weeks. The invasion may be sudden; more frequently it is slow and insidious, after a period in which loss of appetite, dyspepsia, and some muscular weakness have been complained of. After the advent of chilly sensations (for usually there is no rigor), loss of appetite and nausea become marked; headache, backache, prostration, and restlessness, and sometimes insomnia develop, the patient's condition being marked by listlessness or despondency out of proportion to the degree of fever.

The pyrexia is most irregular and variable, the marked irregularity of the temperature curves constituting the chief characteristic of the disease. The fever at the commencement of the attack is not unlike that seen in enteric, being of a

continuous type for several days, with marked evening exacerbations. In mild attacks the temperature may become normal at the end of a fortnight, when convalescence in a small proportion of cases sets in.

As a rule, however, several or numerous relapses occur, in which the temperature undulates in a remarkable manner for a period of ten days to three weeks, the relapses being often separated by but a few apyrexial days. During the relapse-periods the temperature is usually markedly *remittent* in type, and it may even be so from the beginning; thus it may register 100° in the morning and 104° in the evening, and this condition of the chart may continue for more than a fortnight. In other cases, where the temperature in the morning is normal or below it, the evening rise of three or more degrees gives the character of a very distinct *intermittency* to the chart. As the disease pursues its slow course, the apyrexial intervals between the relapses or periods of undulating pyrexia tend to become longer, and the intermitting undulatory periods shorter till convalescence sets in, which may not be for a period of two or three, and sometimes not for six months or longer.

During the whole progress of the malady the nerve-prostration continues to be marked; the listlessness and despondency increase, so that the patient may have little idea of the passage of time or of events about him. Various neuralgic pains set in, and a very constant accompaniment of the late attacks is a severe rheumatic inflammation of numerous joints and of the tendons, and the fibrous structures surrounding them. Orchitis occurs sometimes; the spleen is always enlarged; perspirations and sweat-rashes are constantly present; constipation is almost invariably troublesome and anæmia marked, so that convalescence is very protracted, the entire illness not being recovered from for a period of a year or more. The heart is for a long time quick and irritable; rapidity of pulse may be a marked feature all through the prolonged attack, and bronchial complications frequently occur.

Diagnosis.—The only difficulty lies in the exclusion of *typhoid fever* at first. The absence of the rose-spots, of diarrhoea, and the irregular remittent type of the pyrexia settle the diagnosis at a later stage.

Quotidian, tertian, or other malarial fevers, which are not rare in the same zone, are easily differentiated by the examination of the blood. In cases of much difficulty, puncture of the spleen by a sterilised hypodermic needle will show the micrococcus always present in Malta fever. The existence of the organisms in the urine affords proof of the nature of the disease; the agglutination-test and the determination of the opsonic or phagocytic index may be also employed in the diagnosis. It must be always remembered that, though the agglutination-test is of the greatest value in all

acute cases, the reaction may not be obtainable in the very protracted attacks. The urine is always highly acid.

Prognosis.—Though the disease is so protracted, owing to the number of relapses, the mortality does not exceed 2 per cent. Relapses have been known to occur after many years of latency.

Treatment.—No drug possesses any specific action. The main line of treatment should be that indicated in a prolonged attack of enteric fever; high temperature should be reduced by the use of the cold douche, and symptoms treated as they arise, skilled nursing and a well-regulated milk and light soup diet being of more importance than drugging with anti-pyretics. Wright has obtained excellent results in chronic cases by vaccine-therapy, which is also prophylactic.

MARASMUS

This term is often used to designate the condition of general atrophy or wasting which supervenes in many chronic affections. Though usually the result of organic diseases like cancer, syphilis, tuberculosis, &c., it may be seen in its typical condition in various functional affections, as in the neuroses of the stomach, hysteria, hypochondriasis, &c., where the nutrition of the body is profoundly affected. Though the fatty tissue is the one which suffers most, the muscles and internal organs participate in the general wasting, and are usually the seat of fatty degeneration.

The use of the term 'marasmus' is, however, more intimately associated with the various forms of wasting which are present in infancy.

Infantile marasmus is practically synonymous, in the majority of cases, with starvation. It may, for example, be the mechanical result of some congenital defect in the stomach as pyloric narrowing, or of the mouth as in hare-lip or cleft palate. Improper or unsuitable food may act as a cause of marasmus as effectually almost as the absence of suitable nourishment. In the same manner severe gastric and intestinal catarrh, by preventing the assimilation of nourishment, will eventuate in wasting. Congenital syphilis, by its influence upon metabolism, causes a progressive marasmus in a different way, being often unassociated with faulty digestion, as is sometimes seen in breast-fed infants. These five factors are usually regarded as each the cause of marasmus, but very frequently they are associated together; thus unsuitable food will excite a catarrhal inflammation in the infant's stomach or bowels, which, even after proper feeding, may long remain as a cause of general wasting. Burnet

believes that there is a *primary* marasmus which shows itself, during the first six months of life, by an inherited weakness of the digestive tract in infants whose parents are alcoholic or neurasthenic. It is also, he thinks, often to be met with in prematurely born children.

After the termination of the first year of infant life, improper feeding, as a cause of marasmus, gives way to *mesenteric gland disease* as a factor, there being an intermediate period in which the general wasting is associated most commonly with *rickets*.

Infantile marasmus being thus mainly a product or result of the improper feeding of the infant, the principles of its treatment are obvious. Unless where chills and exposure are permitted to set up catarrhal inflammation of the bowel and stomach in breast-fed children, there are few examples of marasmus met with, except in those fed upon cow's milk and artificial foods.

Undoubtedly the most frequent factor in the production of the general wasting of infants is feeding with cow's milk. The casein of cow's milk coagulates into a firm curd, which is much more difficult of digestion than the flocculent precipitate produced during the first change of human milk in the stomach. Stern considers, however, that it is the chemical nature of the *fat* of cow's milk which is really at fault ; since he finds that it contains much butyric acid, and that double the amount of undigested fat is found in the fæces of an infant fed with cow's milk than in the fæces of one fed upon human milk. Though this may be true to a certain extent, it cannot be doubted that the hard cheesy curds are the main factors in producing the vomiting, catarrh, and diarrhoea which eventuate in infantile marasmus. The improper use of farinaceous food in the early stages of the infant's life is also a potent factor in general wasting. The entire question of the feeding of infants deprived of their natural nourishment through incapacity of the mother is a wide subject beyond the scope of the present brief article. The obvious indication in almost every case is a healthy wet-nurse ; where this cannot be obtained, the milk of the cow should always be given in a state of dilution with weak barley-water. Sterilisation and peptonisation of the milk is, in some cases, indispensable. When the fat of milk is indigestible, egg-yolk may be substituted. For many years the writer has continued to obtain excellent results with the persevering use of the cod-liver oil binder in every form of marasmus occurring in childhood, but especially in mesenteric gland disease.

The marasmus of syphilis, tubercle, rickets, dysentery, gastric neurosis, &c., must be dealt with by the remedies suitable for the treatment of the primary disease.

MEASLES,

Known also as Morbilli, is an acute contagious fever, characterised by symptoms of catarrhal inflammation of the mucous membrane of the eyes, nose, and respiratory apparatus and the appearance of a papular blotchy rash, preceded by characteristic bluish-white spots upon the mucous membrane on the inside of the cheeks, and often by prodromal skin-rashes.

Etiology.—The disease affects subjects of all ages, and both sexes indiscriminately ; but during the first four or six months of life there appears to be a certain degree of immunity. In isolated communities, which have not been protected by previous attacks, the adult and aged members are seriously attacked along with the children, though in all civilised countries the disease is rare in adults, owing to the frequency with which these have already suffered during childhood. As a rule, one attack confers immunity, recurrences being rare.

Though various micro-organisms have been found in the bronchial and nasal secretions, the specific virus has not hitherto been demonstrated ; it cannot be doubted, however, that the microbe exists in these, and is spread through their dissemination in the act of sneezing or coughing. Rosenberger has published recently a description of an oval hyaline body, of the diameter of about one-eighth of a red blood-disc, which he has found almost constantly in the serum of a blister applied to a patient suffering from measles, but he failed to cultivate the organism outside the body. A most important fact has been frequently demonstrated—viz. *that the disease is highly contagious from the very first hours of the invasion, long before the appearance of the eruption.* Though the virus soon loses its virulence, it can be transmitted by fomites adhering to articles of clothing, and it abounds in the *débris* from the cutaneous surface, by which means it is easily conveyed by healthy persons who have been in contact with the patient or in close proximity to the sick-room.

The distribution of measles is world-wide, and, being more or less independent of climate, it is not a seasonal disease ; but Sir John Moore has pointed out that, in Ireland, a mean temperature above 58·6° and below 42° is equally inimical to its prevalence. It is endemic in all cities, and tends to become more or less epidemic, usually in the spring and autumn, when outbreaks are very common.

Symptoms.—The *incubation* period is usually ten days ; in Hebra's inoculation-experiment it was reduced to eight days, but it has been recorded in many cases as extending to eighteen days. In this stage there are no symptoms or evidence of departure from the standard of health.

The invasion is usually abrupt; rarely is it insidious. The child first shows symptoms of catarrh of the eyes, nose, and larynx, sometimes ushered in with a chill, and not infrequently by a convulsion. The temperature rises rapidly, attaining a height often of 102° or 104° before the end of the first twenty-four hours. This *initial* fever, as it is called, sometimes subsides to the normal, or approaches it, before the rash appears, when it again rises.

By the end of the first day the clinical picture is that of a severe feverish catarrh, but, the constitutional symptoms of malaise, headache, and joint-pains being more severe, the aspect is characteristic. There is some suffusion of the conjunctiva, with lacrymation, photophobia, and œdema of the lids. The nasal mucous membrane secretes an irritating fluid which reddens the upper lip; sneezing and hacking cough with hoarseness, and a raw sensation in the throat which makes swallowing and coughing painful, set in simultaneously. The visible mucous membranes are deeply injected, the swelling in the nose usually causing obstruction to breathing; epistaxis is common in some epidemics, the tongue is furred, appetite diminishes or is lost, and thirst is complained of. The cough is always hoarse or croupy, and, when severe, may come in paroxysms like pertussis, and sometimes ends in a fit of laryngismus.

Eruptive Stage.—After the above symptoms have lasted for three days with increasing severity, though the temperature has usually fallen, the *rash appears on the fourth day*. The period of invasion—*i.e.* that between the first onset and the appearance of the characteristic eruption—is thus longer than in the other exanthemata—*viz.* chicken-pox, scarlatina, small-pox, rôtheln, and erysipelas; and it is sometimes extended to the sixth day. The rash is accompanied, or immediately preceded, by a further rise of temperature, great rapidity of pulse, and often by smart diarrhœa. It appears first upon the forehead, spreading rapidly to the cheeks; next upon the back of the wrists and trunk, invading the arms and lower extremities last. Consisting of closely set small red points, which at first disappear upon pressure and are slightly elevated, these rapidly coalesce, forming crescentic patches of a deep crimson or dusky-red colour with sinuous edges, enclosing areas of healthy skin. In many severe cases, however, the eruption becomes confluent, and ecchymoses or miliary vesicles may be observed in various parts of it.

By thirty-six hours the eruption is at its height and the fastigium of the fever is reached—a feature insisted upon by Moore as of vital importance in the differentiation from small-pox. The laryngeal and coryzal symptoms also reach their maximum intensity at this period, and signs of bronchitis are audible all over the chest. The rash now rapidly becomes dusky

and steadily fades, so that only remnants of it are visible as yellow stains within a week of its first appearance. Desquamation occurs with the fading of the rash; usually it is furfuraceous, and begins about the ninth day after the onset, and continues, though scarcely perceptibly, for about a week longer.

Prodromal Rashes.—Considerable attention of late years has been directed to various eruptions which precede the characteristic rash of measles. If the patients be carefully examined, in nearly half the cases, upon the first or second day there will be observed more or less extensive areas of the surface of the trunk occupied by an eruption, macular, papular, erythematous, urticarial, or roseolar in nature, which may be discrete or confluent. Such prodromal rashes are rarely seen on the face and neck; they usually disappear before the true eruption of the disease appears, and the probability of their occurrence and their nature should be carefully kept in mind in order that errors in diagnosis be not committed.

Filatow and Koplik have described a sign which when present is now regarded as pathognomonic of measles. This consists in the presence of small isolated rose-red spots with a bluish-white centre—or, as stated in different words, as white specks, surrounded by a red areola, which are visible on the buccal mucous membrane, often in clusters, opposite the bases of the upper and lower milk-molars, and occasionally upon the inner surface of the lower lip.

The importance of these maculæ, which are known as Filatow's or Koplik's spots, lies in the fact that they are often visible as early as the first or second day of the invasion, frequently before the appearance of the prodromal rashes, and they remain till the true eruption begins to fade. Since they are to be found in nearly every case, and in no other disease, their great value as a diagnostic sign is beyond doubt. Falkener differentiates these spots from stomatitis by the absence of ulceration, by their smaller size, and by the invariable red areola surrounding each white speck.

Upon the fading of the measles-eruption defervescence rapidly sets in; the temperature suddenly falls and reaches the normal usually in twenty-four or thirty-six hours, but the bronchial symptoms do not yield so suddenly; appetite returns, thirst diminishes, and the pulse falls to normal, and by the end of ten or twelve days from the onset the child is practically well.

Relapses are rare, but complications and sequelæ may protract the convalescence.

Variations in the manifestations of the disease from the above type are not uncommon; thus there is undoubtedly a form of measles which is not followed by any eruption, and another with eruption but without catarrh. A *malignant* variety rarely met with is associated with abundant petechiæ

and purpuric hæmorrhages from the mucous surfaces, and is often fatal in a few days. It is also allied to the so-called *black measles*. Adynamic or asthenic types are very common in some epidemics.

Complications.—The catarrh of the eyes, nose, throat, and bronchial tubes cannot be regarded as complications—they are manifestations of the disease; but in some cases the degree of the *laryngitis* may exceed its usual range, ulceration supervening, or even destruction of the cartilages. *Membranous laryngitis* is also frequently met with as a true complication, as in scarlatina, in which case it is produced by the diphtheria bacillus. *Epistaxis* may assume formidable dangers in anæmic children.

Broncho-pneumonia is the most frequent and fatal complication of measles, starting from the bronchial catarrh of the onset-period, which may pass into *suffocative* catarrh at any stage of the disease, or, being reinforced by pneumococcus, streptococcus, or staphylococcus secondary infections, it invades the smaller bronchi and air-vesicles. This broncho-catarrhal or lobular pneumonia usually sets in just after the eruption has reached its height. It is a very common incident in epidemics in the winter, and is liable to follow exposure, sometimes causing the death of half of the children attacked by measles. The popular idea that measles is a much less serious disease than scarlatina leads to the neglect of the patients in working-class districts, where the mortality sometimes becomes appalling. *Lobar pneumonia* is comparatively rare, but at a later stage of the disease a form of tubercular pneumonia, ending in or originating in a *general acute tuberculosis*, is by no means uncommon. *Pertussis* is frequently associated with measles, especially in some epidemics.

Otitis media is not uncommon, laying the foundation of permanent deafness, or, by its extension, causing mastoid disease, meningitis, or fatal cerebral abscess, unless a speedy incision of the tympanic membrane be performed.

Stomatitis of the ulcerative type, originating in the primary catarrh of the mouth, reinforced by staphylococcus infection, is common, and may be followed under bad hygienic surroundings by the fatal gangrenous form, or *cancrum oris*. *Gangrene of the vulva* occurs under similar conditions, and *conjunctivitis* of a purulent type, which sometimes leads to destruction of the eyeball, is occasionally observed; though *phlyctenular ophthalmia* and *keratitis* are more common. *Parotitis* is sometimes met with.

Diarrhœa, which is common and transient at the height of the eruptive stage, may remain, and, increasing in severity, it not rarely proves fatal, owing to acute catarrhal inflammation of the colon.

Kidney-disease is much less common than in scarlatina, but acute desquamative nephritis does very occasionally occur, which may pass into the chronic incurable disease. A slight degree of albuminuria is often observed during the fastigium of the fever.

Heart-complications are rare: when endocarditis or pericarditis shows itself, it is usually in association with *arthritis*; and *chorea*, which is a rare event, may be owing to the same association.

Nerve-diseases complicate measles only in a comparatively small proportion of the cases. As already stated, convulsions not very infrequently usher in the invasion-period in children, and it is in these that a permanent *hemiplegia* sometimes results. *Paraplegia* from acute myelitis is still more unusual, but various types of *paralysis* caused by a multiple peripheral neuritis have been noticed. *Infantile paralysis* and *disseminated myelitis* have been observed, the latter sometimes passing into *disseminated sclerosis*.

Abscess of the brain and *meningitis* have already been referred to as arising from otitis.

Of the above formidable list of complications the pulmonary affections are by far the most serious and frequent, and explain the high mortality of the disease. Some of them may appear as *sequelæ*, and of these tuberculosis or phthisis especially must not be forgotten. Enlargement of the lymphatic glands and tonsils, adenoid growths, and occasionally valvular lesions follow.

Morbid Anatomy.—The changes observed are confined to the congested membrane of the air-passages and to the lymphatic tissue, especially of the bronchial glands, which are nearly always found engorged with blood, softened, and swollen, and the same condition has been observed in the intestinal solitary glands and patches of Peyer. The pathological changes met with as the result of the complications differ in no way from those seen in other diseases.

Diagnosis.—From *scarlatina* the disease is differentiated easily, as a rule, by the lengthened period of invasion (three entire days) before the appearance of the rash, which in scarlatina appears within twenty-four hours after the first symptoms of onset. The lacrymation, coryza, photophobia, and cough are very distinctive in measles, whilst in scarlatina the first symptom is often vomiting. The difficulty is, however, great in those cases of measles where a prodromal rash appears upon the first day, and it is then that the value of Koplik's spots on the buccal membrane is appreciated, their presence being pathognomonic. The deep congestion and swelling of the throat, with the strawberry-tongue and enlargement of the glands, are characteristic in scarlatina. The scarlatina-rash is confluent, consisting of scarlet points, with

the diffuse boiled-lobster redness ; the measles-eruption being papular, blotchy, crescentic, discrete, and more dusky.

Rötheln, when severe, may so closely simulate a mild attack of measles that diagnosis is impossible, since the rashes are almost identical ; but the discovery of the buccal spots is conclusive. The invasion is shorter in *rötheln* (only one to three days), the glandular enlargement is always a very marked feature, the fever is milder, and catarrhal symptoms are always slighter. (See also under 'Scarlatina' for the diagnostic features of the so-called 'Fourth Disease' described by Clement Dukes.)

Small-pox is differentiated by the absence of catarrhal symptoms, the severity of the backache, and the hard shotty feel of the papules, which continue palpable after the skin has been stretched over them—a result never obtainable in measles. A very important diagnostic point will settle the difficulty if the temperature is carefully observed ; this falls markedly in small-pox as the eruption comes out, whilst in measles it continues to rise. In the confluent type of small-pox the period of invasion is shorter, the rash appearing upon the second or third day.

Copaiba produces in some patients an eruption which may be identical with measles, but the absence of all fever is diagnostic ; in the same way the rashes produced by iodides, antipyrin, &c., are easily differentiated.

Prognosis.—This is very good in uncomplicated cases where skilled nursing and proper housing are obtainable, under which circumstances the mortality may not reach 1 per cent. The fever itself is answerable for but a small number of deaths, but the pulmonary complications may actually in some epidemics bring the death-roll up to 50 per cent. The 1875 Fiji Islands outbreak wiped out more than one-fourth part of the population. The prognosis in overcrowded hospitals is grave where isolation of the bronchopneumonic cases is not carefully attended to, since this and other complications are easily spread, being of themselves highly infectious.

Age is an important element in prognosis, the highest mortality occurring between six and twelve months ; the death-rate rapidly falls after the age of two years ; over 90 per cent. of all the deaths occur under five years. Breast-fed infants under six months often escape the disease, or recover with slight symptoms ; the writer has twice witnessed recovery to occur in infants born with the rash out. Pregnancy deepens the prognosis, but not considerably ; the coincidence of whooping-cough in epidemics greatly adds to the mortality. Early fading of the rash is not so serious an occurrence as was formerly believed, but a petechial eruption is unfavourable.

Treatment.—*Prophylaxis* is of vital importance in such a serious disease, but it is surrounded with great difficulty, since the infection is intense long before the appearance of the characteristic rash. Dawson Williams rightly insists upon the necessity of compulsory notification, so often neglected by sanitary authorities, as only by this means can a decision be arrived at for the compulsory closure of infants' schools at the beginning of an epidemic.

A vital point in prophylaxis is the education of the working-class population as regards the mortality at different ages, the parents of children too often acting upon the belief that, as the child must catch the disease, the sooner it gets over an attack the better. Thousands of lives in England would be saved annually if more stringent precautions were exercised in shielding infants and very young children from infection.

A convalescent measles-patient should not be regarded as safe from conveying infection till after the expiration of at least three weeks.

The treatment during the attack must be conducted upon general lines, since no known drug exercises any specific action upon the development of the microbe. Symptoms are to be treated as they arise, the patient being sent to bed in a well-ventilated and carefully warmed room as soon as the first symptoms appear. Uncomplicated cases require little management save judicious milk-feeding and careful nursing, and the avoidance of all exposure to draughts. High temperature rarely, except in the presence of complications, requires active treatment; but it is not a bad plan to adopt the routine administration of a harmless diaphoretic mixture, and, should high fever supervene, cold-sponging may be resorted to. An abundant supply of cold water, acidulated fluid, or diluted milk should be permitted to assuage thirst.

As desquamation sets in, warm baths administered in the sick-room may be freely permitted, and inunction of oil afterwards relieves itching and tends to diminish the spread of the disease.

Complications are to be treated upon the recognised principles suitable to each, as described under 'Broncho-pneumonia,' 'Pneumonia,' &c. Owing to the unhealthy condition of the mouth and the tendency to the growth and rapid development of the germs which cause secondary infections, it is most desirable, and sometimes imperative, that antiseptics should be used from the onset. Since the majority of patients are too young to use these, the best routine method is to employ frequently a weak carbolic spray (1 in 150) in order to cleanse thoroughly the nose, mouth, and throat; by these means laryngeal symptoms are also markedly relieved or prevented, and otitis controlled or rendered impossible.

The convalescent period, though requiring less care than in scarlatina, should be watched assiduously always, in delicate children especially, in the winter months, and premature exposure prevented.

MEASLES, GERMAN

This is a mild specific infectious fever, characterised by a rose-coloured papular eruption, enlargement of the cervical lymphatic glands, and slight catarrh of the throat and sometimes of the eyes. It has been described under several names—viz. Rötheln, Epidemic Rose-rash, Rubella, Rubeola Notha, and formerly as Bastard Measles.

Etiology.—The disease occurs usually in epidemic form; it attacks children of either sex indiscriminately, but adults often suffer, though rarely after the age of forty. It is spread by contagion, and often its epidemics extend over a wide area. As in the other exanthemata, one attack generally confers immunity; but in the writer's experience second and even third attacks are much more common than in the case of measles and scarlatina. Previous attacks of measles or scarlatina confer no immunity from German measles; hence the old idea that it was a hybrid form of these affections is no longer held: in other words, as expressed by Sir John Moore, *Rötheln breeds true*.

Symptoms.—The *incubation* period is usually about fourteen days, but it is liable to considerable variations, being sometimes a week shorter or longer than this.

The *invasion* period is short and variable; thus in many cases the first thing to attract attention is the presence of the rash, there being then, strictly speaking, no duration of the invasion period. More frequently, however, the rash may be said to appear upon the second day after mild feverish symptoms or malaise has set in; these are often accompanied with some sore-throat and a suspicion of coryza or catarrh, but this is never marked as in measles.

The rash appears first about the mouth, and by the next day it has extended over the body and extremities. It consists of rose-coloured points or minute specks, which are so slightly elevated that they may be more correctly classed as maculæ than as papules. These coalesce, but rarely form the crescentic patches seen in measles; but sometimes they may be so universally distributed as to cause a confluent eruption not unlike scarlatina in brightness of colour, being intermediate in this respect between measles and that disease. The general characters of the eruption thus obviously partake of the features of both affections; and this, together with the fact that there is marked diffuse

redness of the fauces and some soreness of the throat, causes great difficulty in the exclusion of scarlatina, and sometimes of measles.

The fever is, as already stated, always mild; rarely is it over 101° . In several cases in an epidemic occurring in a large isolated industrial school, when first seen by the writer, the temperature was normal or but 99° . The state of the pulse, tongue, and other symptoms correspond to the temperature.

Glandular enlargement is a constant sign. The lymphatic glands are usually found tender and indurated at the angle of the jaw, in the posterior cervical and occipital regions, and, in smart cases, in the axilla and inguinal spaces.

Desquamation shows itself as a fine furfuraceous dustiness of the skin, which does not commence for eight days or more, as the rash itself lasts sometimes as long as five or six days, though convalescence may appear to be established by this time.

The disease cannot be said to have any special pathological anatomy; complications or sequelæ, being so rarely met with, may be regarded as coincidents, but slight albuminuria may be noticed occasionally.

Diagnosis.—Under 'Measles' the differential diagnosis has been already discussed. *Scarlatina* has a very short incubation-period, vomiting often is present, and malaise is greater; the temperature is commonly high, nearly always higher than is seen in the smartest case of rōtheln; the pulse is very quick, the enlargement of the lymphatic glands is confined to the neck, usually at the angle of the jaw at first, and never involves the axillary or inguinal group. The nature of the rash is, upon the whole, more scarlatiniform than measly; but often a diagnosis is impossible from the rash alone, and the development of the strawberry-tongue and deep faucial and tonsillar engorgement of scarlatina must be awaited before a positive diagnosis can be made.

Sometimes the diagnosis is quite impossible even after due consideration of every point, and then it is wise to assume that the case is one of scarlatina till the absence in due time of the characteristic peeling or flaky desquamation proves the case to be one of rōtheln.

Prognosis.—Once the diagnosis is positive, the forecast may be pronounced in the same strain—it is always favourable; the child, unless the victim of some other disease, is convalescent in a fortnight, and may be permitted to mix with other children in three weeks from the onset.

Treatment.—This consists in the measures suitable in the treatment of an unusually mild attack of measles.

Some authorities regard the mild examples of the disease as cases differing from rōtheln when the rash comes out with-

out any preceding feverishness and quickly covers the body without being obvious on the face, fading rapidly in twenty-four to thirty-six hours, when marked glandular enlargement and sore-throat cannot be detected. For these cases the name of *Infectious Roseola* has been proposed; and it is stated that they desquamate more freely. (See the description of the so-called 'Fourth Disease' of Clement Dukes, described under the diagnosis of 'Scarlatina.')

MEDIASTINUM, DISEASES OF

The diseased processes met with in this region of the body are usually secondary to inflammatory or other morbid conditions arising in neighbouring tissues or organs, or they are the result of tumours or aneurisms.

MEDIASTINITIS

Etiology and Pathology.—The condition is usually an extension of adhesive inflammation of the pleura or of the pericardium; hence it is frequently styled *Mediastino-pericarditis*. The contents of the anterior mediastinum are found densely indurated or matted together by fibrous tissue; the pericardium is adherent to the epicardium on its inner aspect and united to the pleura and chest-walls on its outer surface. The physical signs and symptoms in chronic cases are those of adherent pericardium, with its great hypertrophy of the heart, cyanosis, dyspnœa, sometimes with the pulsus paradoxus (in which the pulse is lost during each inspiratory period). There may be anasarca of the upper part of the body, and sometimes chronic peritonitis, with ascites and dropsy of the legs, albuminuria, and hepatic enlargement.

Mediastinitis of a suppurative nature, ending in *mediastinal abscess*, has its origin in trauma, tubercular adenitis, erysipelas, typhoid or other fever, actinomycosis, &c. The anterior mediastinum is more commonly involved than the posterior; it is often seen as a sequel to tracheotomy. A primary or so-called idiopathic form of acute suppurative mediastinitis has been occasionally met with which is supposed to be the result of exposure to cold.

Symptoms.—These are variable, but throbbing pain behind the sternum, with severe fever and rigors, is usually present, followed by localised tenderness and œdema; and if the abscess be large, dulness on percussion, fluctuation, and pressure-symptoms may show themselves. The pus is evacuated by the side of the sternum through an intercostal space, or it may find its way into the trachea, œsophagus, or pleural cavity.

ANEURISMS

Occurring in the mediastinum are described under 'Aneurism of the Thoracic Aorta.'

LYMPHADENITIS

Etiology and Pathology.—Inflammation involving the mediastinum is commonly due to tuberculosis of the bronchial and tracheal lymphatic glands, which are often affected in pulmonary phthisis. In children these glands may be the seat of extensive deposit without tubercular affection of the lung-tissue, just as they are often found confined to the cervical or mesenteric regions.

The pathological changes in the glands differ in no respect from those found in other groups of glands or tissue infected by the tubercular bacillus. The gland-substance, however, seems to offer greater resistance to the microbe, so that resolution more frequently follows than when other organs are involved. In the majority of cases, however, caseation follows the production of grey granulations, and suppuration is liable to supervene, leading to chronic mediastinal abscesses. Sometimes, however, the gland undergoes calcareous degeneration after caseation without the formation of pus.

The degree of enlargement of the mediastinal glands (anterior and posterior) necessary for the production of symptoms varies greatly. Often after death very considerable tumours may be found where no suspicion of glandular enlargement existed, and this is perhaps more frequently true of the posterior group.

Symptoms.—These are chiefly, if not entirely, caused by pressure on neighbouring structures. The recurrent laryngeal branch of the vagus may be involved, leading to paralysis of either or both cords, changes in the voice, and cough with the peculiar characters described in thoracic aneurisms—viz. the cough is paroxysmal and accompanied by a brassy clanging quality. Dyspnoea is common, and when the great veins are pressed upon there is oedema of the face and neck. The trachea is usually able to resist pressure, but when the mass of glands is large there may be severe stridor and signs of suffocation.

Dysphagia may result from pressure upon the œsophagus, and even changes in the pupils may be observed. The pleura, and eventually the lung-tissue in the immediate vicinity, by direct extension may become involved in the tubercular process, causing symptoms and signs of pleural effusion and pulmonary phthisis. The most characteristic signs of 'bronchial phthisis,' so-called, occur when the tubercular glands break down and suppurate. The pus usually finds its way into the trachea or large bronchi, and is expectorated with *debris* of

the glands, and blood; in rare instances suffocation and death have resulted in this way before the perforating mass has been expelled by coughing. Less frequently the pus finds its way into the œsophagus or into the pericardial sac, setting up a purulent pericarditis; or it may perforate the aorta or pulmonary artery.

In the chronic indolent enlargements, fever may be entirely absent; but in the suppurative cases pyrexia is present, and usually considerable pain, the clinical picture being obviously that of a suppurative mediastinitis. In rare cases the purulent contents of the gland may inspissate and dry up, or calcification may result. Dulness may be detected over the sternum, more frequently over the upper interscapular region, or at one side of the spine. The auscultatory signs are very variable; there may be absence of breath-sounds over a considerable portion of either lung when the bronchus supplying it is compressed, and bronchial breathing may sometimes be heard in the lung in the immediate vicinity of the mass of enlarged glands. The signs of pulmonary phthisis, when this is present, will, of course, complicate the diagnosis.

Prognosis.—The forecast in tubercular enlargement of the bronchial glands is always serious, but recovery is not infrequent, even after the discharge of pus. The results of *post-mortem* examinations in children dying from various causes show that resolution of old tubercular lymphadenomata is by no means rarely met with, some of the glands being completely calcified. The serious danger is the infection of the lung by the deep bronchial glands.

The treatment is that of the tubercular condition—open air, over-feeding, cod-liver oil, &c.

Mediastinal lymphadenitis of a non-tubercular nature is constantly observed in the infections which produce bronchopneumonia, measles, rōtheln, whooping-cough, &c. There is little tendency to suppuration in these cases, and the symptoms, if any, are masked by the primary disease; the prognosis is always good, and no special treatment is indicated.

TUMOURS

When these are caused by new-growths in the mediastinal space, they are more frequently found in the posterior mediastinum—*i.e.* in that part of the space behind the pericardium containing the posterior mediastinal lymphatic glands; the anterior mediastinum, so often the seat of lymphadenitis, is less frequently involved. The *superior* mediastinum is defined by Symington as bounded below by a plane passing from the lower border of the fourth dorsal vertebra behind to the junction of the manubrium with the body of the sternum in front, its upper limit corresponding with the superior aperture of the thorax. It contains, in addition to the great

veins, trachea, thoracic duct, œsophagus, and nerves, the whole of the arch of the aorta and the thoracic portions of the left common carotid and subclavian; hence its tumours are chiefly aneurismal growths. As, however, it also contains the remains of the thymus gland and the cardiac lymphatic glands, it is also not infrequently the site of new-growths, which may extend up into the neck.

Carcinomata and *Sarcomata* make up the great bulk of the neoplasms found in the mediastinal space; lymphomata, hydatid and dermoid cysts, and gummata are not uncommon, but almost every form of tumour, including the fatty, cartilaginous, and osseous, has been recorded. Their origin is usually in the lymphatic glands, and less frequently in the thymus, but a considerable number originating in the lung and pleura eventually invade the lymphatics of the mediastinal space. The fibrous or osseous tissues are the origin of others.

The etiology of mediastinal growths need not be considered, since as little is known of them as of neoplasms occurring elsewhere. Males are more commonly affected than females. In the rare examples in which trauma figures as a causal factor, the disease may appear at an early age; otherwise the usual period of incidence is about the ages of thirty-five or forty, somewhat later in the secondary cases, which constitute the majority of them, primary sarcoma being more common than primary cancer.

Symptoms.—These will vary somewhat in their general features according to the degree of malignancy or innocence peculiar to the nature of the growth. Thus, cancer will early produce its characteristic emaciation and profound dyscrasia, and run a rapid course—usually within a year from its first appearance; whilst the sarcomata pursue a slower course, and may assume huge proportions before any constitutional symptoms are produced. In other ways an opinion may be formed about the nature of the tumour by observing its tendency to infiltrate neighbouring tissues and by careful search for growths in distant parts.

The symptoms by which mediastinal growths are recognised are in the main, however, those which are due to compression of the various organs, tissues, or vessels in their neighbourhood, though occasionally bulging and other visible evidence of the tumour itself may be obvious in the absence of pressure-symptoms of any kind; but this is rare.

Pressure on the veins produces obstruction more readily than in the case of arteries, and, moreover, the coats of the veins are much more frequently directly involved in the growth, so that thrombosis leading to complete obliteration may affect the superior cava, jugular, and subclavian veins, in which case there will be great œdema and cyanosis of the face, head, and upper extremities, till the appearance of

numerous large cutaneous venous branches show that the blood is finding a return by anastomosis. The obstruction in the pulmonary circuit may cause engorgement accompanied by infarcts, consolidation, and alarming hæmoptysis; blocking of the inferior cava is less frequent. The pressure of large tumours in the superior mediastinum may cause differences in the radial pulse of each side, but this symptom is more characteristic of aneurismal pressure. In posterior and superior mediastinal growths the pressure on the œsophagus may cause great difficulty of swallowing.

Pressure-symptoms caused by interference with the entry of air into the lung are most varied in kind and in degree. The chief of them is dyspnœa; they are described on a previous page (982). The dyspnœa may be paroxysmal and associated with stridor, change in the voice, clanging cough, &c.; it is either the result of compression of the trachea, chief bronchi, recurrent laryngeal nerves, or of direct pressure upon the heart and great veins. As a rule, it has the same character as that met with in aneurismal pressure—viz. there may be no evidence of any difficulty of breathing till the patient makes some slight exertion, as, for example, that produced by dressing or undressing, when a violent paroxysm of dyspnœa or orthopnœa may be brought on associated with marked cyanosis and anxiety. Moreover, these attacks may bear no proportion to the physical signs.

Pressure on the sensory nerve-trunks and their branches will cause more or less pain of a neuralgic character, which may be the first symptom to attract attention; it may be referred to the interscapular region, or to the sternum, or may radiate round the sides of the chest. When erosion of the thoracic walls or spine occurs (which is a much rarer event than in aneurism), the pain may be most excruciating and continuous. Paralysis of the lower extremities may result from spinal erosion, causing injury or destruction of motor nerves.

Fever is seldom present, but occasionally a considerable elevation of temperature has been recorded in rapidly growing tumours during their earlier stages and during the secondary inflammatory involvement of the pleura, or following the lung-changes in infarctions, which cause pneumonic symptoms.

Physical Signs.—The situation and magnitude of the tumour cause these to be as varied as are the subjective symptoms. There may be considerable bulging of one side of the thorax or prominence of the sternum, especially in erosion-cases, and the pulsations of the heart may be transmitted to the swelling, which sometimes may be as pulsatile as an aneurism. When pressure is confined to the root of the lung, its air-supply being cut off, the organ collapses and the affected side falls in, the asymmetry of the thorax

being most puzzling, as the opposite side expands from hypertrophy of the free lung.

Displacement of organs may be observed to an astonishing degree without symptoms of distress of any kind, the heart being thrust over to the right or the liver depressed below the costal arch. When pleural effusion occurs, as it not uncommonly does, its own physical signs are superadded, and these may entirely overshadow the tumour-signs.

The laryngeal mirror may demonstrate pressure upon the recurrent nerves, causing abductor or complete paralysis, and explain cough, voice-change, paroxysmal dyspnoea, &c.

Inspection will also reveal more or less immobility of the chest-wall over the growth, airless lung, or distended pleura.

Dulness on percussion may be elicited over the tumour-region, either behind when the posterior mediastinal glands are the origin of disease, but more frequently in the superior mediastinum, when the tumour can be directly percussed and palpated as it projects above the clavicle and sternum into the neck and infects the cervical glands. The unaffected side is usually hyper-resonant.

Auscultation will reveal the absence of respiratory murmurs over the dull region, and the cardiac sounds are usually not transmitted with distinctness unless the growth is in direct proximity to or in contact with the heart. Vocal fremitus and vocal resonance are usually absent or very much diminished in intensity. Upon the opposite side the breath-sounds are exaggerated, but in the *early* stage these may be intensified upon the affected side before the lung becomes compressed.

The heart's impulse may be visible and the sounds heard with maximum intensity over it, in its displaced position, at a great distance from its normal site. It may be sometimes invisible, and the location of the organ may be difficult when the middle mediastinum is the site of growths which surround, overlap, or entirely conceal the ventricles, especially when pericardial effusion supervenes. Signs of altered innervation of the heart, as determined by the pulse and rhythm, are often present, and feebleness of the ventricular contractions is marked in the advanced stages of cancerous tumours in the mediastinum, as in other sites of this disease.

In addition to the above signs, the cyanosis, œdema, dyspnoea, and enlargement of cutaneous veins over the upper part of the body are also apparent, and clubbing of the fingertips may be sometimes observed at a comparatively early stage.

Diagnosis.—The most important diagnostic points in the complex and puzzling clinical picture of mediastinal tumours have been emphasised in the enumeration of the symptoms and signs. As these are in the main the result of pressure, they

afford little, if any, specific evidence of the nature of the compression which they exercise, and hence *aortic aneurisms* in the same situations are closely simulated. The differential points have already been detailed under 'Aneurism,' upon page 45. These are the absence of a history of syphilis, strain, or obvious arterial changes so constantly met with in aneurism; the marked tendency of the arteries to escape whilst the veins commonly suffer, and the absence of murmurs and of tracheal tugging, are strong characteristic features in tumour-cases.

The history and progress afford considerable assistance in the diagnosis, the short duration and rapid emaciation following the appearance of mediastinal tumours being often marked, as most of the cases run a fatal course within a year, or less. When pulsation occurs in mediastinal growths which have reached the surface, it is never of the characteristic expansile type met with in aneurism, and, moreover, the strong diastolic shock accompanying it is rarely or never present in tumour-cases. The therapeutic test is of considerable value; in aneurism, rest and the effects of large doses of iodides are usually very marked in the relief of pain, which is, moreover, often severer than in tumour. The secondary involvement of the lymphatic cervical glands, when this occurs, is distinctive of malignant growths. The differential diagnosis of enlarged *bronchial glands* is usually not a difficult one: the early age of the patient, the comparative rarity of a high degree of pressure which is more slowly developed, the presence of tubercular glands elsewhere, the frequency with which phthisis complicates the case, and more especially the tendency of the glands to suppurate, are all more or less characteristic.

Prognosis.—This is invariably grave, a fatal issue being the rule inside twelve months, and often sooner in malignant cases.

Treatment.—Very few cases come within the range of surgery, the only treatment available being that suitable for the relief of symptoms as they arise.

MEDULLA OBLONGATA, DISEASES OF

Under 'Paralysis (Bulbar)' are described the affections which are characteristic of degenerative and other changes involving this part of the nervous system, and under other headings are described the affections which are produced by the implication of the nerves which originate in it. Situated between the pons and spinal cord, and in close relations with the cerebellum, lesions of these portions of the central nervous

system also are liable to implicate the medulla, or bulb as it is commonly called.

The following diagnostic points in the localisation of primary bulbar lesions may be mentioned.

Hæmorrhage into the medulla, or fourth ventricle situated in its upper part, is usually rapidly fatal, owing to the injury inflicted upon the respiratory and cardiac centres situated in it. Even when the hæmorrhage is a small one, such a result may be instantaneous. As extravasations into the bulb are generally associated with a similar condition in the pons, it is difficult to be certain of their exact localisation by the symptoms, which are those of a profound unconsciousness, stertorous breathing, and sometimes convulsions, though these latter may be the secondary results of the impeded respiration. Should the hæmorrhage be a very small one, however, and the patient survive the initial unconsciousness, there would be paralysis of those parts which are affected in glosso-labio-laryngeal, or ordinary bulbar paralysis. Sudden death from injury to the medulla is not rare in disease of the atlas and axis and in dislocations of these bones caused by violence, and in head-injuries.

Thrombosis or *Embolism*, followed by softening, often shows itself by severe apoplectiform and sudden symptoms like those of hæmorrhage, but in a considerable number of cases the symptoms are slower in their onset and more chronic in their course, so that localisation may be comparatively easy. There will be paralysis, usually bilateral, of the limbs, sometimes of all four, or of the upper or of the lower extremities only. The characteristic feature is the association with this paresis or paralysis of a similar condition in the parts supplied by one or more of the following nerves—viz. the hypoglossal, glossopharyngeal, and spinal accessory, as evidenced by interference with speech, deglutition, circulation, and respiration. The paralysis of the bulbar nerves, though unequal in degree and extent, is usually bilateral, owing to the close proximity of the centres on each side of the middle line.

When the paralysis of the extremities is unilateral, which is a rare event, the hypoglossal may be affected upon the opposite side, the facial muscles always escaping, except the lips. The bilateral paralysis of the tongue is seldom so marked as in progressive bulbar paralysis, and there is no wasting of the organ; but sometimes complete loss of articulation may be witnessed, owing to the affection of the palate, pharynx, and sometimes of the larynx. This loss of speech (not to be confounded with aphasia), in association with the paralysis of the limbs, pharynx, palate, and larynx, is characteristic of bulbar lesions. Occasionally ataxic symptoms in the limbs have been met with

Bilateral lesions higher up in the cerebrum, it must be borne in mind, will cause symptoms very similar to those of bulbar paralysis; thus, softening of the lower parts of both ascending frontal convolutions will produce bilateral paralysis of the tongue, throat, and lips (pseudo-bulbar paralysis), and bilateral lesions of the internal capsule cause a double hemiplegia, which simulates bulbar disease, but the absence of interference with deglutition, respiration, and cardiac rhythm will distinguish such cases; moreover, true aphasia may be present, which is totally different from the anarthria of bulbar softening.

Another localising sign in lesions of the medulla, in addition to the interference with deglutition, articulation, and respiration, is the alteration in the cardiac rhythm just mentioned; this may show itself by tachycardia, bradycardia, or marked irregularity from involvement of the spinal accessory or pneumogastric centres, and may be associated with laryngeal paralysis or with dyspnœa.

Tumours in the medulla are rarely confined to it, but involve the cerebellum and pons at the same time. The chief localising symptoms may be paralysis of the hypoglossal and spinal accessory.

All the localising symptoms characteristic of bulbar lesions are produced in their typical form in the degeneration, causing progressive glosso-labio-laryngeal paralysis; and it is to be remembered that anæsthesia of the affected parts does not occur; there is little, if any, change in the electrical irritability, though there may be marked wasting in the muscles. Of all the solitary signs met with in bulbar disease, the most characteristic is total paralysis of deglutition, since many of the other symptoms, when isolated, may be caused by lesions in the pons; even partial dysphagia may be caused by pontine lesions, especially when the hypoglossal is paralysed.

MEGRIM, or MIGRAINE

This, also known as Sick Headache, Paroxysmal Headache, Nervous Headache, and Hemicrania, is an affection characterised by irregularly recurring paroxysms of severe headache often unilateral in distribution, usually preceded by visual and other disturbances, and often accompanied by anorexia, nausea, or bilious vomiting, and marked mental depression or irritability.

Etiology.—Hereditary history is usually well-marked in severe cases; thus the writer has had under observation for years a family of six members—the father, mother, and four children—suffering almost constantly from severe typical attacks, whilst these were common in several uncles and

aunts on both sides. A history of other neuroses is commonly associated with the heredity; thus, in the cases just mentioned asthma existed in two, and pseudo-anginal attacks in one member of the family.

The disease is, upon the whole, more frequent in females. It first shows itself in late childhood or after the age of fifteen, and its first appearance after thirty is decidedly unusual. The attacks usually diminish in intensity as age advances, and, becoming less frequent, often entirely disappear in old age.

Amongst exciting causes which are liable to bring on an attack, the most potent is vexation accompanied by excitement; thus a severe attack of 'temper' is almost certain to precipitate paroxysms in a migrainous subject. Any mental over-work, or strain, grief, worry, insomnia, interrupted or disturbed sleep, bodily fatigue, especially in over-heated and ill-ventilated rooms, are all potent factors. Errors in dietary, especially over-feeding, causing a dyspeptic attack, are blamed more frequently than they deserve, as the popular mind associates megrim with biliousness and generally attributes the paroxysms to certain articles of food ingested. It is possible that some of these supposed guilty articles of diet may act by suggestion in neurotic patients. It seems almost certain that constipation has little, if any, influence in inducing an attack; purging, moreover, has no power to ward off or relieve a paroxysm already started. Sedentary occupations or habits are the rule in the migrainous, and the constipation is too often the result of these, and not the cause of the headache.

Eye-strain is undoubtedly the cause of attacks in some subjects, and the correction of astigmatism or other ocular errors is often followed by permanent relief. Severe over-fatigue of a healthy eye may induce a paroxysm, especially in anæmic subjects. Gout has been frequently blamed as a cause of megrim, since attacks of headache of the migrainous type are frequently observed in the children of gouty parents; the association is interesting, being seized upon by those who associate megrim with uric acid and by those who regard the disease as a toxæmia.

Symptoms.—Premonitory symptoms are absent in nearly half the cases, especially in those attacks of headache which begin as soon as the patient awakes in the early morning. When present, the premonitory sensory disturbances may come on with suddenness; they are usually of a visual character. In one case under the writer's care the first attack was ushered in with alarmingly sudden and complete blindness. The individual, in perfect health a moment before, whilst looking at his watch, found himself totally blind, being unable to locate a window in the room through which direct sunshine

was entering. More frequently the eye-disturbance begins as a blind spot near the centre of vision, which spreads over one-half the field, causing hemianopia; or a very bright spot may be noticed instead on one side of the field, or a dimness of vision suddenly affects one-half of each visual field. All degrees and varieties of visual disturbances are experienced; and though the hemianopia may be complete, the loss of sight is very rarely complete, as it was in the case of the first attack just mentioned, where the patient was led back to his house totally blind for half an hour.

The most characteristic of the visual disturbances is that in which, whilst the patient's eyelids are closed, spectral phenomena occur; a luminous spot expands, which becomes dim in the centre, whilst its outer margin shows a zigzag luminous outline with numerous angles, resembling the plan of a fort. This is described as the 'fortification spectrum,' and sometimes its dark parts are illuminated with beautiful lines of colour. Some patients describe the bright zigzag outline as similar to the effect of forked lightning against a dark sky. Though the visual disturbances are often unilateral, they generally affect both eyes in half their visual fields. Other sensory disturbances are observed, as numbness, or 'pins and needles' in the tongue or lips, in one finger, or in any part of the body. There may be a transient aphasia, giddiness, or loss of memory for a few moments, and occasionally slight motor disturbances; but the most marked mental characteristic is a feeling of depression with restlessness, which is quite as common as the ocular phenomena.

The visual disturbances last sometimes for twenty minutes or longer, but rarely exceed half an hour before the advent of the headache. This is of the most varied type and distribution, but usually it is severe. The pain begins very often in a small spot on either temple, or the forehead, or at the back of one or both eyes. Its character is of a boring kind, as if an instrument was being used to gouge out the eyeball or penetrate the bone. When it maintains its unilateral distribution, it may usually be observed as commencing on the side opposite to that in which the visual manifestations were first noted. These pass away as the pain increases steadily in severity, and, as it does so, it spreads over half of the head and may pass over to the other side in some cases. It may commence on the vertex, or occipital region, and may be diffused from the beginning; sometimes it is described as superficial, and at other times as deep. It may radiate to the neck and even pass down the arm of the affected side, as in neuralgia. Usually there is little tenderness, even when the pain has remained confined to a small area, but it is very common to find considerable hyperæsthesia of the scalp next day.

The duration of the pain varies greatly ; it may pass away in an hour, but usually it lasts for several hours, and commonly for a whole day. Migrainous subjects sometimes experience the prodromal sensory disturbances in some attacks without headache or vomiting following ; but this is rare.

As the headache reaches a severe degree of intensity, nausea and vomiting supervene ; but as a rule the emesis is not followed by relief. Nausea and total anorexia are present ; but should the patient yield to the urgent solicitations of her attendants and partake of food, in a few hours this will be rejected almost in the condition in which it was swallowed, the digestive function being completely arrested during the paroxysm. The vomiting soon changes into retching, in which continual attempts are made to bring up stomach-contents, but, the organ being empty, only a little regurgitated bile is ejected, as in sea-sickness ; hence the popular belief that the disorder is the result of biliousness.

Great physical and mental prostration accompany the attack, though marked irritability of the special senses remains. Thus the patient seeks a quiet darkened room ; the introduction of bright light and noises, or the vibrations caused by heavy footfalls, greatly aggravate the intensity of the headache and cause throbbing of the temples.

Vaso-motor disturbances are usually present. Thus the face is often pale and pinched, though the head may feel hot to the touch ; the temporal artery on one or both sides may feel rigid, and the pupils unequal, or one side of the face may be cold and covered with clammy perspiration whilst the opposite side and ear are flushed, dry, and hot. Even when the pain has been confined from the first to the side, vertex, or occipital region, the eyeballs may ache and become tender on the least pressure. The pulse varies : it is often quickened at the early stage of the paroxysm and falls much below the normal later on.

The pain seldom or never stops suddenly unless under the action of a full dose of some analgesic drug. Usually it fades away gradually, till the patient is enabled to fall asleep after several hours of suffering ; when he awakes he finds himself free. In very severe cases the attack may last a couple of days, with brief snatches of unrefreshing sleep.

Though the main feature in *megrism* is usually said to be its characteristic paroxysmal nature, the attacks often showing marked periodicity, yet the intervals are usually of very irregular duration. This is often owing to the migrainous subject escaping for long periods the exciting cause which determines the attack. Some patients, by avoiding mental over-work, irregularity in the hour of retiring to rest, or by avoiding the over-use of the eyes or exposure to bright light, may prolong the intervals almost indefinitely. It is not

unusual for an attack to be precipitated by the termination of the menstrual period, or for the disease entirely to disappear upon the advent of the climacteric.

Even in the most typical cases the different attacks may vary considerably in the same subject, some paroxysms being unaccompanied by vomiting, whilst others may consist only of the prodromal visual or sensory disturbances without the headache. In many cases the intensity of the exciting worry, mental emotion, or other cause may be seen to aggravate the degree and duration of an individual paroxysm.

In some subjects the paroxysmal nature of the attacks may be entirely masked by an almost continuous migrainous headache experienced for long periods upon awaking in the morning, which may lessen greatly in intensity without passing away as evening approaches, or whilst the patient is absorbed in some interesting occupation or amusement. In such subjects, however, a strong exciting cause by precipitating a severe typical attack will demonstrate the nature of the headache, and these patients usually show a marked history of heredity. The continuous pain is often described as the *status hemicranialis*.

Pathology.—The disease has no tendency towards a fatal issue, and cannot be said to possess a pathological anatomy of its own. Various theories have been elaborated to explain the symptoms and to locate the seat of the disturbances observed. The affection has been defined as a paroxysmal neurosis of the epileptic type, caused by the periodic discharge of nerve-force from the sensory centres in the brain. This theory, maintained by Liveing, is sometimes referred to as 'the nerve-storm hypothesis,' and is supposed to explain the connection of true megrim with epilepsy. Gowers published several cases where the one condition had arisen out of or supervened upon the other. Many of the prodromal sensory symptoms of megrim are witnessed occasionally as auras in true epilepsy. J. Mackenzie, in the excellent article in Allbutt's 'System,' labours to show that the phenomena of megrim are the result of excitation of the sympathetic caused by a transient toxæmia. Spasm of the cerebral arteries and dilatation of these vessels have each been maintained as a cause, and have led to the division of megrim into two varieties by German writers—one called *hemicrania sympathetico-spastica*, in which the affected side of the head and face is cold and the pupil dilated, and the other *h. s.-paralytica*, in which the opposite condition is observed. Latham believes that the sensory symptoms are caused by vaso-motor spasm and the headache by dilatation of the vessels.

Gowers maintains that the sensory symptoms are due to a combination of inhibition and of discharge from some deranged action in the cells of the sensory centres, which cannot

be explained by the theory of vaso-motor spasm, the visual disturbance originating in the occipital lobe and the aphasia and the sensory symptoms in the limbs originating in the cortical cells.

A thickening of the arteries on the affected side, as observed by Thoma, is probably the result and not the cause of the affection.

It is interesting to note that in cases marked by transient aphasia the visual symptoms commence in or are confined to the right eye in right-handed subjects as in cerebral hæmorrhage, which goes to prove Gowers' contention of a temporary derangement of the cortical cells of the left frontal convolution present early in the attack.

Diagnosis.—This is easy in all cases in which the visual or sensory disturbances are marked, but the sudden amaurosis of an attack of *uræmia* may be mistaken for migraine. This can be only for a short time, however, as convulsions are almost certain to rapidly follow, and, moreover, the headache precedes or accompanies the eye-symptoms in *uræmia*. Hemicrania by its distribution is almost characteristic when sensory symptoms are absent, and its paroxysmal or recurring nature settles the diagnosis.

The pain of *cerebral tumours* and *organic brain-disease*, though it may be intermittent for *short* periods in rare cases and subject to remissions, is nearly always of a continuous type, and is very usually associated with other symptoms.

Epilepsy can only simulate migraine when it is of the minor type and accompanied by a visual aura, but the momentary duration of the aura is quite characteristic, and in migraine there is never unconsciousness or convulsions.

It must never be forgotten that organic brain-disease may arise in a subject who has suffered all through life from typical migraine, in which case the pain of a cerebral tumour, abscess, or meningitis is very liable to be overlooked; and the headache of renal disease for the same reason may pass unsuspected till other symptoms, as dropsy, &c., supervene.

Prognosis.—This, as regards danger to life, is always certainly favourable; the affection has no tendency towards a fatal issue. Prognosis as regards duration is unfavourably influenced by a strong hereditary history; but in most cases the attacks tend to get less severe as life progresses, and the intervals between them gradually lengthen.

Treatment will favourably influence the duration and severity of the affection, even in the worst cases; and much can be done in the way of preventing attacks by avoiding the exciting causes, especially when astigmatism and over-use of the eyes are at fault.

Treatment.—For the relief of the headache, in many individuals a full dose of any of the newer analgesic drugs

usually brings speedy relief. The agent perhaps most universally satisfactory in its results is antipyrin; but phenacetin, antifebrin, exalgin, and all the other members of the group may be found serviceable. The dose must be a full one; and, since all these agents have some depressing influence upon the heart, the best method will be to administer 5 to 10 grs. citrate of caffeine in combination with the remedy selected. If taken as soon as the first sensory symptoms appear, the headache may be entirely prevented or, in any case, so greatly minimised that the patient may be able to continue his avocation without having to lie down. Unfortunately, the tendency of these drugs in frequently recurring types of the disease is to become less effective as the patient accustoms himself to their use. Purging, emetics, and gastric lavage have their advocates, but probably where good effects have followed their use the case has not been one of megrim.

In the continuous type of megrim relief is not so easily purchased, and resort to drugs of the antipyrin class cannot be recommended, except for the relief of the more severe and acute exacerbations. *Cannabis indica* appears to have some specific action in these chronic examples of megrim, but bromides in full doses afford the best routine method of treatment, and with the last dose of the drug, just before retiring to rest, a small dose of antipyrin may be given; it is a mistake to give caffeine with it, as this drug tends to produce insomnia. Salicylates seem to have some specific action in a few cases, probably when diminished uric-acid elimination or a gouty condition is the exciting or predisposing cause. In very chronic cases, Whitehead's plan of inserting a seton into the tissues over the back of the neck, as described in the author's 'Dictionary of Treatment,' p. 564, is followed by excellent results in most intractable cases. Nitroglycerin is extolled in the hemicrania supposed to be produced by arterial spasm; but little is to be expected from it, save in megrim occurring in chronic renal disease.

The management of the patient between the attacks resolves itself into a scrupulous avoidance of all the known exciting causes, which need not be again enumerated. The dietary should be carefully studied by the physician in each case, and it is a good routine to make the diet as nearly as possible a vegetarian one. Of the general preventive agents, none is so uniformly successful as a total change from sedentary indoor life to an active open-air existence. A prolonged course of arsenic in small doses, combined with a little iron, quinine, and strychnine, is always serviceable.

MELÆNA

This is the name given to the black tar-like motions which are stained by the sulphide of iron derived from the hæmatin of blood poured out high up into the alimentary canal. When blood has escaped from an open vessel in the lower end of the alimentary canal it appears as blood, and usually is easily recognisable by the naked eye as a hæmorrhage. When, however, the lesion is in the stomach, and the partially digested blood is not vomited as 'coffee-grounds,' it passes into the duodenum, and as its hæmogoblin is gradually broken up into hæmatin this is seized upon by the sulphuretted hydrogen in the bowel and converted into iron-sulphide, which, being intimately mixed up with the intestinal secretions and mucus, forms a highly characteristic viscid black treacle-like or tarry motion. When blood is poured out into the pharynx and trickles down the gullet into the stomach the same result is observed. The presence of melæna is, however, most frequently met with in ulcerations of the duodenum and upper part of the small intestine, in which conditions the black motions are seen in their most typical form.

Being the result of the digestive process and the influence of the intestinal gases, their characters are indicative of the time during which the blood has remained in the bowel. Thus the writer has witnessed bright-red arterial blood passed in large quantities after the rupture of a malignant tumour of the œsophagus into the aorta, where the blood was hurried down the canal uninfluenced by the gastric and intestinal secretions.

All shades of colour, from that of the dark-brown hæmatin to the jet-black hue of the sulphide of iron, are met with in melæna, and by careful examination of the stools valuable information may often be gained of the site and extent of the bleeding. In gastric hæmorrhages of considerable degree the extravasated blood is most profoundly changed, being mixed also with mucus, serum, and coagula, which add to the toughness or increased viscosity of the stool. Such stools may consist of nothing but altered blood, food and ordinary feculent material being absent, though tarry motions usually have a fetid odour.

Ulcer of the stomach, simple or cancerous, will generally also betray itself by hæmatemesis, if melæna be also present; the appearance of the latter, if unaccompanied by gastric symptoms, is strong presumptive evidence of duodenal ulceration. A bleeding-point at the lower end of the œsophagus will often be rendered evident by the welling up into the mouth of dark venous blood from varicose veins in this

region, associated with hepatic cirrhosis, but the only evidence may be that of melæna in some cases.

Typhoid, malignant and tubercular ulceration of the small intestine and the upper portion of the large bowel, as well as dysenteric disease, all cause tarry motions. The intense venous congestion of the intestinal tract produced by hepatic cirrhosis, some forms of valvular lesion, and thrombosis of the mesenteric blood-vessels, may cause melæna. The condition also is produced by intestinal parasites, by ulcerative colitis, and by intussusception; it is not a rare event in purpura and other blood-diseases.

Diagnosis.—As a rule, the origin of the hæmorrhage is rendered evident by the symptoms of the disease which has produced it. Thus in typhoid fever the bloody stools commonly met with in the third week cannot be mistaken, in the presence of the fever, diarrhœa, rose-spots, &c.; often sloughs are also present. The history and physical signs of hepatic cirrhosis likewise demonstrate the nature of the melæna. The mucus which is so abundantly present in colitis, dysentery, and intussusception, as well as the other characteristic symptoms of these diseases, prevent mistakes when ordinary care is exercised in their investigation. The greatest difficulty is usually met with in the latent forms of gastric and duodenal ulceration; the diagnostic points are detailed under their respective headings. In all anæmic conditions in which a history of a hæmorrhage cannot be obtained, the evacuations should be carefully inspected, as the patient is frequently unaware of the import of the black tarry or treacle-like stools, and too often attributes these, when his attention has been directed to them, to the action of some medicine which he has been taking.

Iron, bismuth, and manganese produce darkly coloured stools, which, however, are easily differentiated by the experienced eye from melæna. If doubt exists, the motion may be mixed with water, which, upon standing, is not coloured red unless by some blood which has escaped complete digestion. It is worth remembering that patients undergoing a raw meat-juice cure and those partaking freely of spinach may pass stools which simulate melæna.

Where the amount of blood is too trivial to thoroughly blacken the motion in ulceration of the stomach and upper part of the small intestine, by feeding the patient for a day or two upon a milk-diet, and adding a little glacial acetic acid to a watery mixture of a small portion of the motion and shaking it up with ether, the ethereal solution is stained red, as shown by Weber.

As already stated, blood coming from the rectum and lower end of the large intestine maintains its colour, and is recognised by the patient as blood. It is usually, as in the

case of piles, fissure, rectal cancer, &c., associated with the act of defæcation, and is commonly visible upon the surface of the motion, or continues to flow for some time afterwards from the anus. When more intimately mixed with the motion, as in some cases of cancer of the rectum, the tenesmus and mucus which accompany it are characteristic.

The prognosis and treatment of melæna are those of the diseased conditions which produce it.

MELÆNA NEONATORUM

This is a form of hæmorrhage from the bowel occurring in infants from the second to the fifth day of life. The motions may be typical of melæna, or they may be dark-red from the obvious presence of blood poured out low down the intestinal tract. The hæmorrhage is the result of some morbid blood-condition which is probably of microbic origin, since the disease is found oftenest in foundling and lying-in hospitals. There is usually also hæmorrhage from the cord, the nose, mouth, or stomach, or subcutaneous extravasations exist, and not infrequently jaundice is present. The cases are often fatal. Unlike what occurs in hæmophilia, both sexes are affected equally. The *post-mortem* usually reveals no lesion. These serious cases must not be mistaken for the melæna which sometimes occurs when an infant swallows blood sucked from a deeply fissured nipple. They should also be differentiated from the hæmorrhagic form of congenital syphilis, which is characterised by bleeding from the bowel and other mucous surfaces soon after birth.

In the idiopathic form, to which the name 'melæna neonatorum' only should be applied, the temperature is usually elevated; if the infant can be kept alive for a few days recovery spontaneously sets in, as the condition itself is a short-lived one. The best treatment consists in the use of such agents as counteract the shock of the hæmorrhage, as external heat and stimulants.

Chloride of calcium, by increasing the coagulability of the blood, tends to check the hæmorrhages.

MELANÆMIA

This is the name given to the condition of the blood met with in malaria, where the brown or black pigment-granules manufactured by the malarial parasites are showered into the circulation after sporulation. They are taken up by the phagocytes, and may find their way into all the solid viscera, the brain, marrow of bone, and other tissues.

MELANURIA

This word simply means '*black urine.*' The presence of melanin (a sulphur-containing pigment) in the urine is observed in cases of extensive melanotic sarcoma. The urine darkens after standing, and may become as black as ink; usually, however, it only becomes brownish-black, and in rare cases may be of this colour when voided.

The fresh urine may be tested by adding bromine solution, this causes a yellow precipitate which darkens; strong ferric chloride gives to the fresh urine a dark-grey colour, throwing down phosphates and pigment, which are soluble in excess of the reagent. This urine does not reduce Fehling's solution.

Black urine may be the result of *indicanuria* when the secretion contains a large quantity of indoxyl-salt, a substance derived from the indol often present in the intestine, and existing in quantity in cases of obstruction in the small intestine and in cases of putrefaction of proteids in the bowel. The urine becomes greenish-blue upon the surface, and when the specimen stands for a time it may become brownish-black throughout, a condition sometimes observed in typhoid fever, peritonitis, carcinoma, dysenteric conditions, phthisis, &c. The indican can be detected by mixing strong hydrochloric acid and urine together in equal amounts and adding small quantities of solution of chlorinated lime, with continual shaking, when an indigo-blue colour is developed which can be separated by agitation with chloroform.

Alkaptonuria is a condition of the urine characterised by the slow production of a dark-brown or black colour upon exposure to the air; the blackness can be immediately produced on the addition of an alkali, owing to the presence of aromatic bodies formed by some interference with the normal metabolism. The important feature in this variety of blackish urine is that it deeply stains linen and woollen fabrics and reduces Fehling's solution from the presence of dihydroxy-phenyl acetic acid; but it does not ferment with yeast. The condition is often congenital, and remains all through life without apparently injuring the kidney in any way.

Hæmatoporphyrinuria is a commonly recorded form of blackish or dark-red urine, resulting from the presence of iron-free hæmatin, and observed after the prolonged ingestion of sulphonal, and occasionally after intestinal hæmorrhage.

The urine may be coloured almost black from excess of bile as in jaundice, of hæmoglobin as in blackwater fever, and of blood as in hæmaturia; though, strictly speaking, the urine is never really black in these cases, but only of a dark-brown, red, or green. The dark urine of a severe chronic

jaundice case, when diluted with water, will show its yellow or greenish-red tinge. The hæmoglobinuria of the paroxysmal type owes its colour chiefly to methæmoglobin, as do most of the dark urines in renal hæmorrhages. This substance can only be recognised by the spectroscope.

Black urine may be the result of the ingestion of drugs. The following substances stain the urine deeply—viz. carbolic acid, creosote, resorcin, guaiacol, hydroquinone, naphthalin, juniper, sometimes salicylates, and, as already stated, sulphonal, which causes hæmatoporphyrinuria of a deep port-wine tinge. (See also under 'Hæmatoporphyrinuria,' p. 518.)

The orange-red urine caused by the administration of senna, rhubarb, and chrysophanic acid is never so dark as to warrant the use of the name 'melanuria'; it is only changed to a dark red by alkalies.

MENIÈRE'S DISEASE. (See under VERTIGO.)

MENINGES, CEREBRAL, INFLAMMATION OF

The three membranous coverings of the brain—the dura mater, arachnoid, and pia mater—are liable to inflammations of different kinds, and each may also become the source of hæmorrhages.

Inflammation of the outer covering—the dura mater—is usually styled Pachymeningitis; that of the inner coverings—the arachnoid and pia mater—which are always affected conjointly, is known as Leptomeningitis, or inflammation of the pia-arachnoid, or more usually as Meningitis.

Pachymeningitis.—The dense fibrous tissue constituting the cerebral dura mater consists of two layers, the outer of which serves as an internal periosteum to the bones of the cranium. The inner layer is thinner, and is lined upon its internal surface by a smooth layer of endothelium, which covers over an extensive network of large capillaries surrounded by numerous lymph or perivascular channels. These channels, according to Ford Robertson, convey the lymph or cerebro-spinal fluid into the venous system through the cranial veins.

Inflammation may attack either the outer or inner of these two layers of the dura; in the former case it is termed *Pachymeningitis externa*, and in the latter *P. interna*.

EXTERNAL PACHYMENINGITIS

Etiology.—This form of pachymeningitis is more commonly met with than the internal variety, and almost always it is the result of direct injury or disease of the cranial bones,

and hence it is chiefly of surgical interest, occurring either in association with fractures or necrosis of syphilitic origin. It is not rare in middle-ear disease, which causes caries of the petrous bone. In fracture of the flat bones, hæmorrhage between the outer layer of the dura mater and the inner table usually follows the injury, the inflammatory process supervening later. In compound fractures, in erysipelas of the scalp, and in septic scalp-wounds, the inflammatory action is liable to end in suppuration, an abscess being formed between the dura and the bone; very rarely is the location of the abscess between the layers of the membrane where the perivascular channels are situated, though the arachnoid has sometimes been invaded.

Where suppuration does not supervene, there may be considerable thickening of the dura, which becomes glued to the bone by the inflammatory exudate; in this latter eventually ossification may set in.

Symptoms.—These vary with the nature of the primary disease or injury; in any case, headache is liable to be severe and persistent, though more or less localised, and usually associated with tenderness of the scalp. Delirium and rigors, with smart irregular exacerbations of fever, usually mark the advent of suppuration; whilst in the non-purulent cases the temperature may remain normal. Convulsions may occur; in the chronic cases ending in much thickening, these may be unilateral or of the traumatic epileptic type. The extra-dural or sub-cranial abscess, if situated over the motor area, may cause paralysis of a limb or limbs upon the opposite side.

Treatment.—Where the regional diagnosis is certain and the presence of pus suspected, the only procedure available is to evacuate the abscess by trephining; similar treatment may be indicated in the non-suppurative or adhesive type of the disease where intense and persistent headache or unilateral convulsions are marked.

INTERNAL PACHYMENINGITIS

The internal layer of the dura mater is very rarely the seat of a true primary inflammation, but two forms are usually described—viz. the purulent and the hæmorrhagic.

The *purulent* variety is almost invariably secondary to a similar condition in the pia mater and arachnoid, or it may be caused in rare cases by an extension inwards of an inflammation of the outer layer of the dura mater. Osler mentions a third variety, which he has observed in pneumonia, under the title of *pseudo-membranous*.

Both these conditions are of little clinical interest, since their special symptoms, if any such exist, are completely overshadowed by those of the primary affection. The so-

called hæmorrhagic variety of internal cerebral pachymeningitis, however, requires a brief notice.

INTERNAL HÆMORRHAGIC PACHYMEINGITIS

This diseased condition is also known as Hæmatoma of the Dura Mater, Meningeal Blood-tumour, and Subdural False Membrane.

Morbid Anatomy.—In this condition the inner surface of the dura mater is lined by a delicate vascular membrane, which readily peels off, and rarely adheres to the arachnoid. In some cases the vascular membrane seems to be replaced by a layer of laminated blood-clot, whilst in others there is nothing observable but a thin layer of apparently recently coagulated blood. This covering is seen to envelop a considerable proportion of the surface of the convolutions on one or both sides, and there is usually an appreciable amount of atrophy to be noticed in the convolutions beneath it.

Pathology.—Virchow's theory of the pathology of this strange condition is that which is usually accepted. He maintained that it was the result of an *inflammatory* action commencing upon the endothelial surface of the dura, which poured out a layer of fibrinous exudation into which new vessels penetrated and granulation-tissue formed, so that a vascular organised membrane was produced. Owing to the proneness of the newly formed vessels to rupture, hæmorrhages occur, and these are sometimes so extensive as to destroy or disintegrate the membrane, which may disappear, finally leaving nothing but a laminated layer of blood-clot. In the rare cases where hæmorrhages do not take place, a fibrinous sheet of considerable thickness, with its rich vascular network, remains. By others the pathology of the affection is believed to be that of a simple subdural hæmorrhage originating in the veins of the pia mater. Robertson maintains that the process is essentially a *degenerative* and not an inflammatory one, starting in the endothelial layer of the dura by altered nutrition, the result of cerebro-spinal fluid laden with the products of perverted metabolism from the underlying cerebral matter.

Etiology.—The affection is a rare one, except in the insane, being usually met with in the chronic insanity of aged subjects, in general paralysis, and in long-standing cases of dementia and melancholia.

It is also met with in the wasted brains of subjects who have died from chronic phthisis and various cachectic blood-conditions, chronic alcoholism, and after fevers, scurvy, and rickets. Males are much more frequently affected than females.

Symptoms.—The original malady usually masks the symptoms of this form of meningitis, and special clinical

features do not, as a rule, appear till a short time before death. These are commonly apoplecticiform seizures, ushered in with severe headache, vomiting, and signs of increased intracranial pressure, as optic neuritis, slow irregular pulse, sluggishly acting contracted pupils, cerebral vomiting, &c. When the hæmorrhage is unilateral there may be hemiplegic signs of cortical type; convulsions are not frequent, but somnolency, passing into coma, may be the only symptom when the affection is bilateral and extensive.

Diagnosis.—From the vagueness of the symptoms, as just described, it is not surprising that many cases remain undiagnosed till the *post-mortem* examination reveals the true nature of the affection. The existence of the condition should be always suspected in general paralysis of the insane, or in any chronic form of insanity, when a sudden increase in the mental failure or cerebral depression is associated with severe pain over the vertex and the development of signs of intracranial pressure, with great muscular weakness.

Treatment.—All agents which are indicated in hæmorrhage in other regions are useless; trephining can but effect the most temporary relief, and is therefore usually unjustifiable. The malady is perhaps always fatal, though Gowers mentions one alcoholic case in which recovery occurred, as proved by an autopsy six months afterwards, in which sufficient remains of the disease existed to justify the diagnosis.

MENINGITIS, ACUTE CEREBRAL, or ACUTE LEPTOMENINGITIS

This title is applied to an acute inflammation of the pia-arachnoid membrane. The cerebral and spinal meninges being continuous, the latter is generally more or less involved with the former, and, moreover, the inflammation of the cerebral pia mater usually to a certain extent affects the cortex, so that the condition is sometimes described as a meningo-encephalitis.

The disease may be regarded as always the result of the invasion of the cerebral membranes by micro-organisms; hence various types have been recognised. The best-marked of these varieties or types are seen in the meningitis produced by the pus-forming microbes, by the bacilli of tuberculosis, and of cerebro-spinal fever. Though the symptoms and signs of the resulting meningitis are to a large extent identical in most cases, nevertheless, by their degrees of intensity their duration, and other clinical features, it is often easily possible to effect a differentiation in the diagnosis of the specific microbes. The following forms of acute cerebral leptomeningitis will be described—viz. Acute Purulent

Meningitis, Acute Posterior Basic Meningitis of Childhood, Epidemic Cerebro-spinal Meningitis, and Tubercular Meningitis.

MENINGITIS, ACUTE PURULENT, or ACUTE SUPPURATIVE LEPTOMENINGITIS

Etiology.—This condition is frequently found associated with injuries to the skull, which by breaches of continuity in the scalp or bones permit of the introduction of microbes. Erysipelas or purulent affections of the scalp may involve the membranes through the veins of the diploë. Adjacent disease of bone causes the largest number of cases; thus purulent otitis media, caries of the petrous bone, suppuration of the mastoid cells, diseases of the nasal cavities, frontal sinus, orbit, or pharynx, may supply the germs which excite the inflammation. Abscesses of the brain or cerebral tumours, and occasionally an intracranial hæmorrhage, may supply the necessary foci.

The germs may be brought to the meninges from a considerable distance by the blood-stream in septicæmia, pyæmia, ulcerative endocarditis, &c. The meningitis may be the result of an acute general infective process, as in pneumonia, small-pox, typhoid or other fever. Sunstroke, excessive mental emotion, chronic renal disease, and blows upon the head which produce no wound, have been regarded as etiological factors, but they probably only act as exciting or accessory causes by depressing the natural resistance of the meninges to micro-organisms accidentally introduced into the blood-stream.

Morbid Anatomy.—The convexity of the brain is much more frequently the site of purulent meningitis than is the base, the latter being seldom affected alone, unless when the disease is secondary to ear-trouble. The first change is hyperæmia and cloudy swelling of the pia mater; pus appears probably within forty-eight hours over the sulci in isolated foci, which combine and cover the convolutions with a thick layer of purulent secretion spread out under the arachnoid. The superficial stratum of brain-matter is involved in the inflammatory mischief, which also extends a variable distance down the membranes of the spinal cord.

The ventricles of the brain are usually found to contain some clear serous or opaque fibrino-serous fluid, but very rarely pure pus, except in a small percentage of cases occurring in young children. The disease is so rapidly fatal, as a rule, that there is seldom time for such marked ventricular distension as would warrant the name of ‘internal hydrocephalus’; moreover, there is only likely to be real ventricular distension

in meningitis which affects the base and closes the iter, preventing the escape of fluid from the lateral and third ventricles into the subarachnoid spaces. The most typical cases of purulent meningitis of the convexity, or, as they are styled, *vertical meningitis*, are seen associated with acute pneumonia, pericarditis, or ulcerative endocarditis, in which the layer of pus may entirely cover over the convolutions on both sides of the brain and extend downwards towards the base. When the disease is secondary to ear-mischief, it is basal, and may remain unilateral and localised.

Symptoms.—The symptoms in this form of meningitis are liable to be entirely overshadowed by those of the primary affection, so that the diagnosis is often only made after opening the cranial cavity in the routine of a *post-mortem* examination. Headache is usually always present, and if this has been a clinical feature of the primary disease it becomes more marked, persistent, and excruciating, and liable to severe exacerbations, which continue all through the delirium which is also usually present. This persistence of headache during delirium, and even whilst the patient is in a somnolent condition bordering on coma, is a very characteristic feature of purulent meningitis, seldom absent except in those cases arising during pyæmia.

Vomiting is nearly always present, but is not so markedly severe and persistent in the vertical cases as in the basal form. Hyperæsthesia, as evidenced by the slightest movement or by pressure on the scalp, and intolerance of noise and bright light are generally present; the increase of tactile sensibility may be generally distributed or unilateral, and the superficial reflexes are increased. Fever is almost invariably well marked, and during the rigors which are common a hyperpyretic temperature is often reached; not infrequently the fever falls below the normal before death. The pulse is usually quickened, though at the beginning of the attack it may be markedly slow, even when the temperature may register 105°; towards the termination it becomes usually very rapid, weak, and irregular. Convulsions of the epileptic type are not uncommon; they may be preceded by muscular twitchings or local clonic spasms, and seldom remain unilateral, though, upon the whole, they are less frequently observed in purulent meningitis than in the tubercular variety.

Retraction of the head from rigidity of the muscles of the back of the neck is never marked so long as the meningeal mischief is confined to the vertex; its supervention indicates the extension of the disease to the meninges of the posterior basal fossa. Rigidity in other muscles can usually be demonstrated, the abdominal wall being retracted and greatly increased in its resistance to pressure; but this is not seen in basilar meningitis. The entire back-muscles may be

strongly contracted when the inflammatory action extends down the spinal canal.

Kernig's sign is well marked in all cases, and hence of great importance ; it is elicited, as the patient lies upon his back, by flexing the thigh to a right-angle with the body, when it will be found impossible to extend the leg, which may even remain at a right-angle at the knee, even when great force is employed to extend it in line with the femur, so long as the thigh is flexed upon the trunk.

Paralysis of nerves is not a marked clinical feature in purulent vertical nor even in purulent basilar meningitis ; whilst in the non-purulent basilar forms, strabismus, ptosis, and other evidences of loss of power in the cranial nerves are constantly met with. These will be referred to in the description of tubercular and posterior basic meningitis. Hemiplegia may be produced when the meningeal trouble concentrates its action upon the central convolutions of the cortex of the brain, inhibiting their function by irritation and not by direct pressure of the purulent secretion. Monoplegia may be induced in the same manner. The facial nerve is sometimes paralysed by meningitis of the vertical type, but then only in its lower portion, in contradistinction to basilar trouble, which paralyses all parts of the nerve.

The pupils are often contracted in the early stage of the disease, especially when the photophobia is marked ; later on they usually are dilated. Of more importance is marked irregularity of the pupils, one being contracted which in a few hours afterwards may be widely dilated, its fellow sometimes contracting as the dilatation becomes marked.

Optic neuritis is rare in vertical and less infrequent in basilar meningitis, and common in the tubercular form. Aphasia is also a very unusual symptom in purulent vertical meningitis, but vertigo is often met with at the commencement of the attack.

The respiration towards the end is generally most irregular, quick, and superficial, or sighing, resembling in the main the Cheyne-Stokes breathing.

The disturbance of the vaso-motor system shows itself in the *tache cérébrale*, and in the tendency towards bed-sores and sloughing of parts subjected to pressure or to the influence of hot poultices. The knee-reflex, exaggerated at first, is usually absent or diminished as the patient passes into the comatose condition, in which the bladder and intestinal contents may be passed involuntarily, though constipation and retention of urine are the rule in the earlier somnolent stages.

Death may occur before the third day ; usually a fatal issue is not postponed much beyond a week.

Diagnosis.—The presence of a primary disease likely to give rise to purulent meningitis, the rapid onset of intense

headache, delirium, cerebral vomiting, convulsions, and other signs of cortical irritation, together with the short duration of the fatal malady, are usually sufficient to establish the diagnosis of purulent inflammation of the meninges of the convexity of the brain. In *posterior basic meningitis*, which is usually not a purulent inflammation, the slower onset and longer duration, with the more intense vomiting, marked retraction of the head, and often-present signs of ocular paralysis, and *tonic* spasm of the limbs, are sufficient to differentiate the vertical type of the disease. Moreover, basic meningitis arising independently of ear-mischief occurs generally inside the first year of life, and delirium does not occur in its early stages.

Cerebral abscess, which (with sinus-thrombosis) is a not infrequent *complication* of vertical meningitis, may closely simulate the inflammation of the meninges when this latter exists alone. The diagnosis may be almost impossible, since cerebral abscess is so often associated with head-injuries and ear-mischief, which are common causes of purulent meningitis; the slower progress of the case, and the fall in the temperature which often occurs in abscess after the intensity of the acute symptoms has passed away, may assist in the diagnosis.

Pneumonia, typhoid fever, influenza, and various other acute infective and septic diseases must be differentiated by a careful search for their special clinical features, especially as these affections are commonly ushered in with convulsions in the cases occurring in childhood. It must be remembered that these diseases are also liable to be the cause of the meningeal trouble.

Suppurative otitis without any meningeal complication is difficult of exclusion, especially when occurring in children in whom convulsions and delirium are common symptoms; the progress and duration of the disease alone will settle the diagnosis. The presence of optic neuritis is not to be relied upon as a distinctive sign, since it may occur in both affections.

It must be remembered that purulent meningitis, as already stated, may be entirely latent, or only manifest itself by very ambiguous symptoms, and this is most likely to occur in septicæmic or pyæmic cases. In such circumstances *lumbar puncture* may prove a most valuable aid to diagnosis. The space between two lumbar vertebræ, preferably the third and fourth, is made tense by flexing the spine as the patient lies upon his left side, and the skin is pierced in the middle line or slightly to the side of a spinous process by a hollow needle, which is driven upwards and in a median direction till the subarachnoid space is reached. The examination of the opaque cerebro-spinal fluid which usually flows out under increased pressure in purulent meningitis will reveal

pus-cells or leucocytes in great numbers, and the characteristic micro-organisms—streptococci, staphylococci, or pneumococci—may be detected by means of culture-experiments. Where blocking of the iter has occurred, the result may, however, be negative; the discovery of tubercle bacilli and of the germs which cause cerebro-spinal fever will settle the diagnosis of these affections.

Prognosis.—With the establishment of the diagnosis the hope of a favourable prognosis vanishes, as the disease must be regarded as almost invariably fatal.

Treatment.—The only hope of influencing favourably the mortality of the disease lies in prophylactic measures in those cases of head-injury, extradural abscess, and purulent otitis which are likely to be followed by purulent meningitis. Trephining of the mastoid cells, incision of the tympanum, and the evacuation of pus in the vicinity of skull-fractures may entirely prevent the supervention of a purulent meningitis, which would be otherwise inevitable and fatal.

When the purulent inflammation has already extended to the meninges, little can be hoped for except the relief of symptoms. The measures suitable in treatment of tubercular meningitis should be resorted to, as rest, the ice-cap, mercurial purges, leeching, &c. When the disease is confined to the convexity, the evacuation of pus by trephining and incising the dura mater has in a few cases of head-injury and ear-disease been followed by success. The tympanum and mastoid cells should be opened promptly, owing to the difficulty in diagnosing between otitis and vertical meningitis when there is evidence of the former affection, even though the latter is believed to also exist.

Puncture of the ventricles is not indicated in purulent meningitis, but the evacuation of pus by lumbar puncture has been apparently followed by relief in several cases.

MENINGITIS, ACUTE SEROUS.

(See under HYDROCEPHALUS.)

MENINGITIS, ACUTE SIMPLE, OF CHILDHOOD

This form of leptomeningitis has been exhaustively dealt with by Lees and Barlow in their brilliant article in Allbutt's 'System,' under the title of 'Posterior Basic (Occlusive) Meningitis,' in which the details of nearly one hundred cases are analysed. More recently, by the researches of Hunter, Carr, and Wall, the subject has been further illuminated.

Etiology and Pathology.—Simple posterior basic meningitis is a disease mainly of the first year of life, and is extremely rare after the age of four years, if all cases obviously belonging to cerebro-spinal fever be excluded. It is a seasonal disease, inasmuch as it is more common in winter. The problem of the causation of this disease has led to laborious and patient research by a host of investigators, and the outcome of general results goes to prove that the 'posterior meningitis of children is not an isolated condition, but merely a special form of acute cerebro-spinal meningitis' (Carr). The cases which occur in infancy may be regarded as sporadic examples of the disease, and every intermediate form is seen, from the chronic type met with in infancy, which chiefly involves the meninges of the posterior fossa, to the acutely fatal epidemic cases in adults, in which the entire convexity may be affected. In every case the view is held as correct that the disease is the result of the invasion of the meninges by Weichselbaum's *diplococcus intracellularis*, which is found in the cerebro-spinal fluid. Netter and others maintain that certain epidemics of cerebro-spinal meningitis are caused by the *pneumococcus*, the symptoms being identical with those caused by the *D. intracellularis*, and the view is held that the latter microbe may be an attenuated form of the pneumococcus. Wall concludes, after an exhaustive survey of the entire field of discussion—(1) That infection by the diplococcus intracellularis has been shown to be associated with a train of symptoms known by the names of 'epidemic cerebro-spinal meningitis,' 'cervical opisthotonos,' and 'posterior basic meningitis.' (2) That these conditions are therefore identical in their etiology. (3) That cases of chronic hydrocephalus sometimes are consequent upon this form of acute meningitis.

No direct contagion can be traced in this variety of meningitis; there is no evidence that the disease is conveyed from one person to another, the microbe being supposed to gain its entrance through the nasopharynx or tympanum.

The question of the identity of the micro-organism which produces simple posterior basic meningitis with that of the microbe causing epidemic cerebro-spinal fever has been recently raised in a most interesting form by the careful researches of Dr. Thomas Houston and Dr. Rankin. These observers have demonstrated what they consider to be radical differences between the causal organisms in these two diseases. Their researches will be mentioned in the discussion of the bacteriology of epidemic cerebro-spinal meningitis.

Symptoms.—The symptoms already described as occurring in purulent or vertical meningitis are met with—viz. headache, vomiting, fever, convulsions, delirium, and altered or Cheyne-Stokes respiration, &c.

The most important and characteristic sign of the disease is *retraction of the head*, which sets in as early as the first day of the disease in about half the cases, and almost invariably shows itself before the end of the sixth day, and lasts for several weeks in the cases ending in recovery, and in the fatal cases till death.

This symptom is caused by the tonic contraction of the posterior neck-muscles. Marked rigidity may be present without much retraction in some cases. Wall describes this rigidity as resistance to forward flexion of the head. It may be taken as evidence of the irritation of the meninges in the neighbourhood of the pons and medulla; hence its very great value as showing the posterior basilar nature of the attack. It occurs in tubercular meningitis, but this, if it becomes basic, generally starts in the anterior fossa, consequently retraction of the head, when present, is a late phenomenon, and is never so strongly marked as it is in posterior basic meningitis, of which it often is the first sign. Owing to the prominence of the head-retraction, posterior basic meningitis was described in 1878 by Gee and Barlow as 'cervical opisthotonos.'

Distension of the fontanelle is an early symptom; at a late stage it is indicative of acquired hydrocephalus.

Ocular symptoms are met with—thus strabismus is present in nearly half the cases, but it is not usually due to paralysis affecting a particular nerve; a certain degree of staring is common from retraction of the upper lids, and nystagmus is not rare. The pupils are contracted at first; somewhat later they may be unequal, and towards the end they are dilated. Amaurosis often occurs, though optic neuritis is comparatively rare, and even when total blindness is present the pupils react, though sluggishly, to light; the amaurosis is central, and may disappear after recovery.

Contractions of the jaw and lip-muscles, causing biting or sucking movements and grinding of the teeth, are very common.

Tonic spasm of various muscles is regarded by Barlow as a very characteristic sign; it is a similar manifestation to opisthotonos and to the rigidity which is so constant in the neck-muscles. The extensors usually suffer most, though the lower limbs may be flexed whilst the upper are extended, and *vice versa*. This is regarded as the result of irritation of the cerebellum.

Convulsions of a clonic nature are not uncommon, and are indicative of irritation spreading to the motor cortex. Definite paralysis of any nerve or nerves is rarely seen, the predominating symptoms being irritative rather than paralytic. Screaming is very commonly present, especially in the earlier stages, though there is little evidence of hyperæsthesia, and a severe crying-fit often brings on an attack of tonic

spasms resembling the condition seen in tetanus ; sometimes a fit of clonic convulsions may accompany it.

The pulse, fever, and respiration do not very markedly differ from what is observable in tubercular meningitis ; usually in the chronic cases the temperature falls and remains sub-normal.

An interesting feature is the rarity of skin-eruptions, considering the unquestionably close relation of simple basic meningitis to the cerebro-spinal fever of older subjects ; occasionally fleeting erythematous patches appear in different regions, but herpes, according to Lees and Barlow, is very rare.

Wasting is a marked feature in all cases which do not succumb early ; it is not associated with the obvious retraction of the abdomen seen in purulent and tubercular meningitis. Vomiting is usually more severe and persistent than in these affections.

Joint-complications, as evidenced by local heat, redness, and swelling, which usually entirely disappear, are occasionally seen, but not so often as in epidemic cerebro-spinal meningitis.

The most frequent result of posterior basic meningitis is the production of distension of the cerebral ventricles, causing hydrocephalus. This is held by Lees and Barlow to be owing to the mechanical interference with the escape 'of the fluid from the lateral and third ventricles through the iter, the fourth ventricle and its three openings into the posterior subarachnoid space, and so into the meshwork of subarachnoid tissue extending under the base of the brain and into the large subarachnoid space surrounding the spinal cord in its whole length' ; and these authorities maintain that in almost every case where hydrocephalus follows, the autopsy will reveal some definite obstruction in this drainage-channel of the brain or cord. Wall still adheres to the inflammatory theory, and Carr thinks that the last word has not been said upon the subject. Though the low s.g. of and the absence of albumin in the cerebro-spinal fluid are in favour of the mechanical theory as opposed to the inflammatory hypothesis, nevertheless the fact that lumbar puncture can so often reduce the intracranial pressure is against the theory of an obstruction in the foramen of Majendie, in the aqueduct of Sylvius, or in the upper part of the spinal canal.

Morbid Anatomy.—This shows the results of a usually non-purulent inflammation, with exudation of lymph in the posterior fossa invading the meninges over the medulla and between it and the cerebellum, and advancing forwards to the optic chiasma. The involvement of the inferior surface of the cerebellum and the contiguous aspect of the medulla explains readily the blocking of the drainage-channel.

Simple basic meningitis often runs a slow course of many weeks, and sometimes of months. Whilst about 15 per cent. recover completely, the remainder either die during the period of acute symptoms or afterwards succumb from the acquired hydrocephalus, or show permanent deafness, blindness, mental weakness, speech-defects, or motor disabilities.

Diagnosis.—The chief points in the differential diagnosis between posterior simple basic meningitis and the *purulent* and *tubercular* varieties have been already emphasised, and will be again referred to under the latter disease. The combination of symptoms of head-retraction, tonic muscular spasm, and the more common meningeal symptoms coming on suddenly in an infant under one year, running a course of several weeks, and ending in temporary blindness and hydrocephalus, renders the diagnosis certain. From *cerebro-spinal meningitis* the condition sometimes cannot be differentiated, and the disease is, as already stated, according to the opinion of most authorities, but a modified sporadic form of the latter-named affection, occurring in children of six to twelve months, and running a much slower course, without the usual rashes observed in the adult cases. The examination of the cerebro-spinal fluid obtained by lumbar puncture will, after staining and culture-experiments, exhibit the slightly modified diplococcus intracellularis of the epidemic form of the disease.

Quincke's serous ventricular meningitis is differentiated by the characters of this affection described under 'Hydrocephalus.'

Prognosis.—The outlook is always grave, but since one in every six to eight cases recovers completely, it is much better than in vertical and tubercular meningitis. The prognosis of the acquired congenital hydrocephalus is discussed under 'Hydrocephalus.'

Treatment.—This is to be carried out on the lines laid down for the management of the other forms of meningeal inflammation—absolute rest, the application of ice to the scalp, and the exhibition of such remedies as are useful in allaying pain, vomiting, and other symptoms. In one important respect the treatment differs from that indicated in the other forms of meningitis—*i.e.* inasmuch as the disease runs a slow course and there is some prospect of recovery even after the expiration of several weeks, feeding should be scrupulously attended to in order to avert a fatal marasmus. With this object, rectal and nasal alimentation should be pushed.

Mercury and iodides give good results in many chronic cases, and they should always have a trial in every instance. Surgical measures are only indicated in the late stages when hydrocephalic symptoms show themselves, and of these lumbar puncture occasionally affords marked temporary

relief. Incision of the tympanic membrane on both sides can do no harm, and should otitis be present the relief may be permanent. Trephining of the occipital bone and drainage of the fourth ventricle seldom does good. Drainage of the lateral ventricles gives better prospects of cure, and if this is made permanent by establishing an opening through the cortex so that the fluid may be absorbed by the arachnoid villi, as accomplished by Sutherland and Watson Cheyne, permanent recovery may result. (See also under 'Hydrocephalus.')

MENINGITIS, CEREBRAL, CHRONIC

As frequently observed, acute cerebro-spinal fever and posterior basic meningitis may run a very slow course of many months; but, strictly speaking, in such instances the symptoms remaining in the chronic stage are not those of meningitis, but of the acquired hydrocephalus, which has resulted from the glueing together of membranous tissues about the foramina and posterior lymph-cistern, or from the chronic inflammatory changes in the ventricular lining. These cases, when first coming under observation after many months, are liable to be wrongly regarded as of syphilitic origin. Chronic meningitis may exist as a localised affection in those cases where a mass of caseating tubercle remains more or less quiescent in some part of the cortex without a general meningeal infection occurring along the vessels supplying the cerebral tissue or membranes. A similar condition may exist in association with other cerebral tumours, or with diseases of the cranial bones.

A general chronic meningitis has been described as occasionally occurring during the course of *chronic alcoholism*. The changes in this form consist in thickening and opacity of the pia-arachnoid, usually over the convexity, such as are observed in general paralysis of the insane, chronic dementia, &c.; and they are regarded by some authorities as of the nature of a degeneration rather than of true inflammatory origin. This form of meningitis is more of pathological than of clinical interest, since it is usually first discovered on the *post-mortem* table.

MENINGITIS, CEREBRAL, CHRONIC, OF SYPHILITIC ORIGIN (SYPHILIS OF THE BRAIN)

Morbid Anatomy.—This shows gummata and arterial changes. The disease is described as *basal gummatous meningitis*, and originates in the subarachnoid tissue about the

chiasma and interpeduncular space, extending along the Sylvian fissures, and finally invading the entire base of the brain, involving the optic and the nerves supplying the eye-muscles, but especially the third nerve. The starting-point may be a localised gumma or a whole series of these springing from the pia mater, or it may begin as a diffused inflammatory cellular new-growth, consisting of vascular granulation-tissue. More frequently this superficial diffuse neoplasm is associated with the presence of the gummatous growths. The gummata vary in size from that of a large pea to a small plum; they are grey and translucent; on section their centres show the fibro-caseous or dry degeneration. Gummata in the cranium almost invariably spring from the membranes, sometimes from the dura mater, secondary meningeal inflammation commencing afterwards in their vicinity. The diffuse superficial neoplasm associated with the tumour-formation, at first of gelatinous consistence, tends to become fibrous and dense, so that the entire base is invaded by a firm layer of new-growth, which extends along the coats of the blood-vessels and the sheaths of the ocular nerves, matting together all the cerebral membranes for a variable extent in the direction of the vertex.

The arteries are diseased, nodular growths projecting from their coats are visible, and these are liable to caseate or soften; the lumen of the vessels finally becomes diminished or obliterated, and hence the tendency to softening of the cerebral tissue at various spots.

The meningeal inflammation involves the superficial parts of the brain in its vicinity, so that the secondary result is a specific or *syphilitic meningo-encephalitis*, which may lead to deep destructive softening in a large area of the brain. Finally, sclerotic lesions may supervene.

The above-mentioned changes constitute the usual pathological picture of so-called 'Syphilis of the Brain.' In a small number of cases the gummatous meningitis may be confined to the convexity; but usually when this region is involved the base of the brain suffers also, and not infrequently the meninges of the spine become the seat of a similar lesion.

Symptoms.—These come on at a variable period after infection, usually within two or three years; they are rarely produced by the congenital disease. In some cases syphilitic meningitis both of cerebral and spinal distribution may be met with during the secondary stage of the acquired disorder. It occasionally appears to be precipitated by traumata.

Though the symptoms vary greatly in intensity and even in kind, basal gummatous meningitis is not usually very difficult of recognition. Most of the signs and symptoms have been already dealt with under 'Cerebral Tumour' and 'Arteritis.'

There is a stage of variable duration at first in which the symptoms are more or less *general*, after which *local* signs show themselves. Headache is the most marked and constantly present forerunner of the manifestations of cerebral basilar syphilis. It is intense, and, though often of constant type, it is very liable to nocturnal exacerbations, the dread of which sometimes sets up severe insomnia. The headache may be frontal, parietal, or occipital, or it may vary from a general headache to a localised one, in which case some tenderness of the scalp may be elicited on light pressure. Vertigo, vomiting, and some departure from the ordinary character of the patient, as evidenced by mental apathy, transient failures of memory, or by attacks of passing confusion of thought or of apparent stupidity, or a condition suggestive of commencing dementia, melancholia, or delusional insanity, may be observed. Slight aphasic symptoms, or inability to call up the proper name or expression, are common prodromata. Epileptic convulsions or passing attacks of excitement or delirium, which may alternate with somnolency and stupor, are not rare.

Local symptoms or signs may suddenly supervene upon any of the above conditions. Paralytic signs are most variable and numerous. If these be the result of the vascular changes, they may be identical with the hemiplegia, bulbar paralysis, and aphasia which are met with in general arterio-sclerosis. When the disease of the middle cerebral or of the basilar artery which produces these symptoms has only led to a moderate degree of occlusion of their calibres the attacks are usually incomplete and transient, passing off sometimes in a few hours or minutes—a feature highly characteristic of syphilitic arteritis. Extensive interference with the patency of the vessels produces permanent paralytic and aphasic symptoms in no way differing from those resulting from softening of the brain-tissue produced by other diseases in the vessels or their branches. The slight hemiplegic attacks, when transient, are almost certain to return and become permanent at a later stage, becoming more progressive and extensive after each attack. Such attacks are usually preceded by severe headache and by the other premonitory symptoms already mentioned; they are not, as a rule, accompanied by unconsciousness.

Hemiplegia is often upon the side *opposite* to that on which the cranial nerves are paralysed, since the vessels are usually more deeply involved on the side where the basilar meningitis is most intense; the hemiplegia may involve both sides when the syphilitic arteritis is extensive.

Ocular palsies are more characteristic of basal syphilitic meningitis, being the direct result of the changes caused by the gummata or diffuse neoplasm at the base of the brain

which implicate vision and involve the motor nerves of the eyeball. The injury to the optic nerves may cause total and permanent blindness. More frequently this is incomplete, bitemporal hemianopsia being more common; or this may exist in one eye with blindness in the other. Atrophy of the disc, or choked disc, may exist on one or both sides; the optic neuritis occurring in the case of syphilitic gummatous meningitis appears early, and is usually rapid in its progress.

The oculo-motor nerves embedded in the growth may cause a complete ophthalmoplegia, though this is comparatively rare; more frequently one or more branches of the third are affected, the fourth and sixth suffering less frequently. Often one muscle after another shows signs of weakness, and when both eyes are involved the paralysis is usually much more marked on the one side than on the other. Ptosis is very commonly present, with or without loss of accommodation.

The olfactory on one side often suffers, causing anosmia. When the fifth becomes involved it is generally unilaterally, and often in association with the seventh or with the sixth of the same side. Its injury causes loss of sensation in the face and cornea, preceded by neuralgic pain and neuro-paralytic keratitis. The seventh nerve when paralysed is often associated with deafness from implication of the auditory on the same side. The paralysis is of the peripheral type, and may be double. With the extension backwards of the syphilitic infiltration paralysis of the bulbar nerves is produced, and when this is limited to one side and found in association with eye-symptoms the nature of the case is obvious. Implication of the spinal meninges is not infrequently observed, and will be referred to under its own heading.

The chief clinical features in all the above palsies are their remarkable *variability, their liability to come and go, and for the paralysis of one nerve to be associated with or to be replaced by that of another within a period sometimes of a few days or weeks.* A facial paralysis on the right side to-day may be unnoticeable to-morrow, or may be on the left side next day. Similar variability is observable in the field of vision, the bitemporal hemianopsia disappearing and in a few days or months again returning. These remissions and exacerbations are independent of the vascular changes (arteritis, &c.) which are liable to cause hemiplegia, aphasia, &c. They are the result of the varying alterations which occur in the syphilitic neoplasm determining oscillations in the pressure exercised upon the embedded nerves.

A solitary gumma springing from the cerebral membranes, as already described under 'Cerebral Tumours,' will produce the ordinary symptoms of any other brain-neoplasm, varying as these do with its localisation, magnitude, rate of growth,

&c. Thus headache, vomiting, optic neuritis, convulsions (general or unilateral), will usually be present when the cortex is involved, and temporary paralysis not infrequently follows the convulsion, or may occur independently of it. Aphasia, though more characteristic of the arterial lesions so constantly present in brain-syphilis, may be also produced by the presence of a tumour. The convulsions appear in some instances to develop into ordinary epilepsy, which may continue after the dissipation of the syphilitic growth by suitable treatment. This circumscribed form of brain-syphilis may occur without the typical basilar meningeal characters already described which are so characteristic of the disease, but these are often present in combination with the localised cortical irritation.

The prolonged torpor or stupor which is liable to come on during the progress of basilar gummatous meningitis may last for two or three weeks, and is highly characteristic. Mental symptoms of deeper gravity than those noticed in the prodromal stage are common, and may pass into the condition sometimes described as *syphilitic dementia* or *pseudo-general paralysis of the insane*, in which grandiose conceptions and the characteristic tremor are, however, usually absent. This dementia paralytica may not, however, proceed to a fatal issue, as is always the case in true general paralysis. As pointed out by Turner, the mental symptoms are caused by the diffuse meningeal and vascular changes, and may in some cases develop into true general paralysis.

The duration of the disease may be said to be almost indefinite in many cases ; sometimes it runs a sub-acute course, but more frequently the headache precedes the advent of local symptoms for many months or years, in which case the onset of paralysis, &c., may partake of an acute character for a time, followed by remissions.

Diagnosis.—The various points in the diagnosis have been already emphasised in the description of the variable and varying symptoms and in their multiform groupings, an essential feature being the history of the primary local lesion and of the secondary phenomena. To these specific characters must be added the amenability to treatment which is almost distinctive of cerebral syphilis when compared with other organic brain-lesions.

Prognosis.—Though always somewhat serious, the outlook in many cases is distinctly hopeful. It is usually very good when the patient is seen in the early stage and the symptoms have supervened in the late secondary period, especially if he has chanced not to have been treated by mercury, as there is then every likelihood of his recent syphilitic neoplastic growths rapidly yielding to a vigorous exhibition of the drug. At the stage when fibroid or sclerotic changes

are liable to be present the prognosis is less favourable, but even then iodides may accomplish much. Vascular changes, which always supervene in the late stages, decidedly justify a grave prognosis, as these must be regarded as practically beyond the range of treatment; hence the removal of a hemiplegia, when not of localised cortical origin and if of any duration, must be looked upon as almost hopeless, though life may still be prolonged for an indefinite period. Recovery may be complete even after optic neuritis has appeared, but marked optic atrophy seldom, if ever, yields.

Localised cortical lesions causing meningitis give a better outlook than diffuse cortical mischief, though even this may ultimately yield to treatment. Many patients recover partially with more or less inaiming, but relapses are always liable to occur.

Treatment.—This in no way differs from the management of syphilitic lesions elsewhere, save that, owing to the dangers liable to result from the pressure of the specific neoplastic growths upon the delicate tissue of the brain and cerebral nerves, the treatment must be pushed with vigour and no time should be lost. Inunction by mercury should therefore be commenced without delay, about 1 dr. of the B.P. ointment being rubbed in daily after dilution with an equal weight of lard, and this treatment should be continued for at least two months, during which time about 8 oz. of the ointment will be necessary. No definite amount can be safely recommended, however, since patients vary so much in their susceptibility. It is a good plan to keep the inunctions up to the limit of touching the gums slightly without producing salivation. In urgent cases Bacelli's intravenous method of injecting 15 mins. of a 1 per cent. solution of cyanide of mercury is justifiable at the commencement of treatment. The inunctions should be repeated every six or twelve months, or as soon as signs of relapse appear. In the intervals iodides should be administered vigorously, and, notwithstanding the cautions emphasised by many authorities, iodide of sodium may be given in 30 gr. doses thrice daily for many months. Mercury and iodides may be advantageously combined in many cases, and sometimes undoubtedly better results are obtained by the conjoint use of the natural baths and inunctions as practised at Aachen by Tyson. (See the author's 'Dictionary of Treatment,' 4th ed. pp. 916-930, where the details of syphilitic treatment are described.)

It is needless to say that in most cases of long duration, where the symptoms are mainly those produced by the vascular changes, mercury, if at all used, must be employed with great caution; but in these instances iodides may be given in large doses for long periods.

MENINGITIS, CEREBRAL, TUBERCULAR

This is an acute inflammation of the pia-arachnoid membrane, especially in the neighbourhood of the base of the brain, commonly at its anterior aspect, caused by the invasion of the bacillus of tubercle. The term 'tubercular leptomeningitis' is sometimes used.

Etiology.—The general causation is that of tuberculosis, and here only the special etiological features need be referred to. Like all the forms of meningitis, it is much more common in children; though it may be met with at any age, it is very rare in advanced life. During the first six months of life it is so rare as to justify the assumption that the nature of a meningeal inflammation is more likely to prove to be of the posterior basie than of the tubercular variety. About one-third of the cases occurs in children between one and two, and another third occurs in those between two and five years of age.

The disease is rarely primary, generally starting from some tubercular focus in the intestine, bones, or lungs, or in the mesenteric or other lymphatic glands. Its frequent occurrence in children reared upon cow's milk, and the known liability of this food to tubercular infection from disease in the cattle supplying it, render it highly probable that in the majority of cases this is the origin. The writer once has seen tubercular meningitis in a young infant fed entirely on the milk of a perfectly healthy mother; so that it is just possible that the bacilli may occasionally find their entrance to the body by the respiratory tract, from which they may invade bronchial or other glands.

Tubercular meningitis is often observed in older subjects to follow scraping, eurenting, or other incomplete operation on a distant tubercular focus. Blows upon the head may determine the disease to the meninges, as so frequently is observed in the case of testicular tuberculosis following a contusion of the scrotum. In such cases the disease originates primarily in the diseased area owing to the diminished resistance of the injured tissue to any bacilli finding their way into the body by the air or food. This is probably the explanation of the frequency with which tubercular meningitis and phthisis follow diseases like measles, pertussis, &c.

Morbid anatomy.—From the frequency with which the disease originates in or spends its force upon the membranes at the anterior base of the brain, it is sometimes described as 'basilar meningitis'; but it must not be confounded with posterior basie meningitis. The frequency with which the ventricles are found distended by fluid gave rise to the old name of Acute Hydrocephalus.

The pia-arachnoid, especially in the vicinity of the interpeduncular space, the optic chiasma, and the Sylvian fissures, is opaque, thickened, and matted. A tough fibrinous or fibrino-purulent exudate of a yellow or greenish colour covers the membrane in these regions, and grey tubercles are observed scattered over the meninges, extending into the covering of the lower surface of the frontal and temporo-sphenoidal lobes and median fissure; but the convexity of the cerebral hemispheres usually escapes inflammatory exudation, though granulations often exist there. The superior vermiform process of the cerebellum has generally its pia-arachnoid infiltrated with tubercles. The choroid plexuses of the lateral ventricles and the velum interpositum often show abundant grey or whitish granulations. The underlying brain-tissue is softened, and presents the usual changes observed in meningo-encephalitis.

The cerebral arteries are always found with their coats invaded by miliary nodules, and many of them show thrombotic changes blocking their calibre; this is well seen in the branches of the middle cerebral and in the arteries of the anterior perforated space, and accounts for the spots of cerebral softening often observed.

The lateral ventricles are distended with fluid and, though true pus is seldom found, the contents are always more or less opaque or turbid from the inflammatory condition of the ependyma. The tension of the ventricular fluid is usually sufficient to cause more or less flattening out of the cerebral convolutions; but the foramina are not blocked, as in the acquired hydrocephalus which follows the posterior basic meningitis.

The amount of tubercular deposit varies greatly, and usually bears no relation to the extent of the inflammatory action. Frequently the grey granulations are found in greatest abundance in the Sylvian fissure, but not rarely a tubercular caseous mass may be found situated in the cortex and forming a tumour of considerable dimensions. In some cases the nodules are so small and few in number that most diligent search is necessary before the nature of the case becomes obvious. Sometimes the meninges of the cord suffer in the tubercular invasion, but rarely to any great extent.

Symptoms.—Where the meningeal invasion is but a part of a general acute tuberculosis supervening upon advanced disease of lungs, bone, or mesenteric glands, the constitutional symptoms may entirely overshadow the meningeal signs; and sometimes in these cases the *post-mortem* examination reveals cerebral mischief not suspected during life.

As the disease in its typical form is most commonly met with in young children, the physician has usually to recognise

its presence only by objective symptoms or signs; and as these vary within very wide limits, it is difficult to include in a brief description a fairly typical case. The onset is usually insidious, being preceded for a variable period of several days or weeks by symptoms of general ill-health.

During this prodromal period there is some loss of appetite, irritability, listlessness, or drowsiness; sleep is often disturbed by startings, and occasionally there is grinding of the teeth or gums. The mother notices that the child is failing without obvious cause and that his natural disposition is altered. If this period is prolonged, marked wasting may be present; constipation is the rule, and occasionally vomiting may be observed. In older children who can describe their sensations occasional headaches are complained of during the prodromal period; frowning, ebullitions of temper, and fretfulness are common, and there may be some aberration noticed in speech, or inability or disinclination to master simple lessons which formerly were a source of pleasure. Mental inertia and marked change in the individual's behaviour suggestive of the onset of insanity may occur during the prodromal stage in adults, whilst the temperature is normal.

The real invasion often is ushered in by a convulsion, which may be general or unilateral; much more frequently it is bilateral and of the usual epileptic character, and it is preceded and followed by intense headache of a most persistent type. In infants the presence of the headache may be inferred from the child placing its hand upon the scalp and screaming continuously or at short intervals. This so-called cephalic cry is almost characteristic, and is commonly recognised by the nurse or parent who has witnessed the progress of a previous case. The child's head is bored backwards into the pillow, and any movement or change of posture seems to aggravate the cephalalgia, and usually brings on a screaming fit.

The temperature will generally be found elevated, but not markedly so, and occasionally it may never rise beyond 100°; its range and fluctuations are in no way distinctive, unless tubercular lesions elsewhere are present.

Vomiting is an early and important sign; it is of the true cerebral type, bears little or no relation to food, and appears to afford no relief. It may continue throughout the attack, but usually its persistency and intensity vary considerably during the late stage. Delirium is present more frequently in adults than in children, and may in them simulate delirium tremens.

After the supervention of the above irritative symptoms the clinical picture is fairly characteristic in children. The patient lies in bed, usually with the decubitus constantly on

the left or right side, with the legs flexed on the abdomen, the arms flexed at the elbows, and the face averted from the light, as photophobia is usually present; a loud noise or vibration brings on a screaming fit. The physiognomy is somewhat characteristic: the features are often fixed in a frown or anxious fretful expression, the face somewhat suffused, the pupils, at first contracted, afterwards dilate, and the eyes seem fixed on a distant object, so that the nurse often reports that the child has become blind; but amaurosis does not occur at this stage. The *tache cérébrale*, so common in many other cerebral conditions, is easily elicited by drawing a line with the finger-nail on the skin. The mental state exhibits marked peevishness, and an outburst of screaming usually follows any attempt at examination of pupils, pulse, or temperature; when the bedclothes are turned down the child very constantly seizes them and pulls them up again. There is often some rigidity of the neck-muscles, but the marked retraction of the head characteristic of posterior basic meningitis is rarely seen. Tonic spasms of the muscles of the face, back, or limbs, of transient duration, are not uncommon, and muscular twitchings or general convulsions like the one which marked the initial onset may occur; but the clonic convulsions of the irritative stage tend towards great variability, being often confined to a limb or to one side of the face or body, as in cortical epilepsy, according to the localisation of the cortical irritation. Tremors are often observed affecting the hands and head or limbs; they are coarse in degree, and are often associated with purposeless muscular movements. Later, rigidity of one or more limbs may be observed, which, though it may vary in intensity, tends to remain till the advent of the final stage. The anterior fontanelle is usually tense, even at an early stage, and pressure upon it causes pain, usually before the stage of intra-ventricular pressure is reached. The pulse is at first rapid, but at this stage becomes irregular and slows somewhat, and the respiration is normal. Kernig's sign is present early.

As the stage of *pressure*, caused by distension of the ventricles, sets in, the peevishness, screaming, headache, and vomiting diminish in intensity, and the somnolence increases, or delirium may show itself for the first time; but consciousness is seldom entirely lost. The abdomen becomes boat-shaped, the muscular walls flaccid, and constipation is obstinate. The tonic or clonic spasms, or even the general convulsions of the irritative stage, may continue at irregular intervals all through the attack. The pulse is usually much slowed and irregular, changing sometimes from hour to hour, and the temperature fluctuates irregularly, frequently sinking below the normal and rising to 102° or more within an hour or two. Slight variability in the respira-

tion-rate may be noticed at this stage; it is often quickened somewhat, but sighing respiration is not uncommon. Erythematous redness may appear upon any region of the cutaneous surface, generally spreading from some part subjected to pressure. The pupils, which remained contracted during the irritative stage, begin to dilate as the intracranial pressure increases; some inequality is also usually observed. If a bright light be held before the eyes, the pupil contracts for a few seconds, but dilates again, and contraction and dilatation may alternately be observed for some minutes. At this stage strabismus and ptosis may appear; sometimes they are noticed early in the irritative period; irregular movements of the globes, to and fro or upwards and downwards, appear, and may simulate nystagmus; signs of paralysis of the facial nerve may be observed, but these, with the ocular palsies, may disappear and recur from time to time during this stage, especially after a convulsion.

Finally, the last or *paralytic* stage is reached, in which the patient, turning from the lateral decubitus, lies upon his back, sinks low down in the bed with his arms and legs extended and the hands crossed and lying over the hypogastric region, his eyes and cheeks being sunken and his body wasted. The pupils are widely dilated, and if they react to light at all the contraction is very slow; one lid is often open, owing to facial paralysis, and the conjunctival reflex is absent as sensation is lost in the cornea. Squint and ptosis are usually present, and slow independent movements of the eyeballs may occur; and, owing to the lids failing to completely close, a film of pus mixed with mucus accumulates upon the surface of the globe; ulceration of the lower corneal margin is not uncommon, as the eyeballs are frequently found turned upwards. The ophthalmoscope reveals optic neuritis, and tubercles in the choroid can be sometimes demonstrated.

The patient steadily sinks into a state of coma, from which at first he may be aroused by the application of a brisk stimulus, but gradually the coma deepens, and complete insensibility generally is present before death. The knee-jerks, which are little altered in the former stages, are now found frequently absent, and the cremasteric reflex usually disappears.

Convulsions, which may have been absent after the beginning of the irritation-stage, not infrequently return; and if so they are bilateral. Though the clonic and tonic spasms or rigidity may remain in one or more limbs for a time, the progress of the case is towards a general paralytic condition; before this becomes complete, paralysis of one limb or hemiplegia may be obvious. During this condition of impending death sometimes a striking remission occurs in which transient consciousness appears, only to pass into deeper coma and

complete insensibility to all stimuli. The power of swallowing is lost; the pulse becomes extremely rapid, though often regular, and it may register 200 or more. The sighing respiration witnessed sometimes in the pressure-stage passes into the Cheyne-Stokes type, and finally the breathing gets very shallow and quick.

The temperature towards the termination often reaches a hyperpyretic height or falls as low as 85° immediately before death. Incontinence of urine and fæces may be present from the onset of the irritative stage, or they may only show themselves during the final paralytic period; occasionally slight albuminuria is observed. The blood may show some leucocytosis, and marked amœboid activity has been described as a characteristic feature by Bastian.

Course and Duration.—In the above description an attempt has been made to enumerate the symptoms and signs in the four typical periods of the disease—viz. in the *premonitory*, *irritative*, *pressure*, and *paralytic* stages; but it must be remembered that these stages constantly overlap each other, so that the features of one stage may be present during the succeeding period, and in some cases there may be no evidence of intra-ventricular pressure at any period of the illness. The duration of each stage is most irregular and variable. The duration of the attack from the onset of the initial convulsion, headache, and vomiting usually lasts for a period under three weeks; more frequently the disease proves fatal within fourteen days, the stage of irritation usually being marked for about seven days. Acute cases are not uncommonly met with which terminate within three or four days. In older children and in adults many symptoms may remain latent, only obstinate vomiting and intense headache being present before the appearance of a severe convulsion, which may be soon followed by a profound and rapidly fatal coma. In these cases there is usually no dilatation of the pupils; probably the meningitis mainly involves the cortex, though few inflammatory changes are to be found after death. When a localised mass of caseating tubercle exists in the cortex the symptoms may resemble those of cerebral tumour, till general meningeal inflammation sets in with the secondary involvement of the blood-vessels by the bacilli, or it may continue as a circumscribed meningo-encephalitis, producing hemiplegia, aphasia, or other focal signs.

Diagnosis.—This is often most difficult in the early stage, and the disease is frequently diagnosed when it does not exist.

Typhoid fever in children is liable to be mistaken for tubercular meningitis, but vomiting is less marked; diarrhœa and the rose-spot eruption may be present. The abdomen in typhoid is usually somewhat distended, never 'carinated'

or boat-shaped, with flaccid walls, as in meningitis ; and the spleen is enlarged. The patient from the onset of the fever generally lies upon his back, and rarely screams or pulls back the bedclothes (Stocker's sign) when uncovered ; the temperature is continuously high, and in children a slow pulse is very exceptional. The presence of Widal's reaction is a valuable aid, and Kernig's sign is absent. At a later stage the wide dilatation of the pupil, strabismus, tonic spasms, and continuance of the severe headache are characteristic of the tubercular disease ; this latter symptom disappears in typhoid when delirium sets in. The detection of tubercles in the choroid is a valuable indication in any doubtful case of tubercular meningitis.

Acute pneumonia in children is differentiated by the suddenness of its onset during a period of perfect health ; it, however, often begins with a convulsion and vomiting, but the pyrexia is very high, the pulse and respiration are much quickened, the skin burning hot, and the eyes bright. The careful exploration of the chest after the first day should be conclusive in pneumonia ; and Kernig's sign is absent.

Tetanus may be simulated, but severe headache, vomiting, and photophobia are not present in tetanus ; the tonic spasms or rigidity of meningitis are not of prolonged duration, trismus is rare, opisthotonos is practically never seen, and the supervention of eye-paralysis and other ocular signs settles the diagnosis of the tubercular affection.

Otitis media in children, especially when double, is likely to be mistaken for tubercular meningitis, the more so when it follows after some other affection, as measles, whooping-cough, &c., in which the tubercular mischief is also likely to appear. The symptoms may be identical, as convulsions, headache, vomiting, delirium, and retraction of the head or rigidity of the neck-muscles, the only difference being observable in the absence of the markedly retracted abdomen and persistent constipation. The appearance of otorrhœa or the removal of pus by freely incising the tympanum may alone clear up the diagnosis. In such cases, should meningitis follow the ear-trouble it is likely to be of the suppurative type ; but not infrequently tubercular meningitis is found in association with old otorrhœa.

Cerebral abscess is most difficult of exclusion ; the scrutiny of the history and previous symptoms, and the discovery of a cause, as bone-disease, ear-trouble, &c., will aid in the diagnosis. Rigors and high temperature are in favour of abscess ; but often the temperature in both conditions may be for a time normal.

Thrombosis of cerebral sinuses closely simulates tubercular meningitis in some cases ; and here again the minute examination of the history of the case is of more importance than

the symptoms present when the case is first brought under notice. The discovery of a local cause or septic focus, as evidenced in petrous bone-disease, pulmonary or hepatic pyæmia, &c., the presence of venous engorgement and œdema about the scalp, with hæmorrhages into the fundus, are in favour of the thrombotic affection.

Posterior basic meningitis is identified by the marked retraction of the head and persistent rigidity or severe tonic spasm of other muscles. Though these occur also in the tubercular form, they are but slight, and often variable or evanescent. The patient is usually under nine months of age, paralysis of cranial nerves is very rare, the slowing of the pulse is less marked, nystagmus and amaurosis are more frequently present, though optic neuritis is oftener met with in the tubercular form. The duration of the attack may in the long run settle the diagnosis, as the disease may drag on for many months; whilst the tubercular form proves fatal inside three weeks, and never exhibits the unmistakable signs of acquired hydrocephalus and great ventricular distension so commonly present in the posterior basic disease.

From *cerebro-spinal fever* the diagnosis is differentiated upon the lines just mentioned, since this affection may be in children practically regarded as posterior basic meningitis.

Suppurative or simple vertical meningitis is distinguished by the usual presence of some septic disease, as pneumonia, pyæmia, empyema, &c., or by the discovery of the source of a local invasion of pus-forming organisms in diseased cranial bones or scalp. This disease is usually fatal in a few days, without ocular symptoms showing themselves, and often with little vomiting; marked general convulsions and very high fever are generally present.

The differential diagnosis of the various forms of meningeal inflammation may often be an impossibility in the early stages without resorting to lumbar puncture. The discovery of the tubercle bacillus in the cerebro-spinal fluid is pathognomonic of tubercular meningitis, but its absence cannot be regarded as conclusive evidence against the disease. An important detail in the investigation of the fluid obtained from a lumbar puncture in tubercular meningitis should not be overlooked. If the fluid be allowed to stand for some time a small clot generally forms. Entangled in the meshes of this clot the bacilli of tubercle can often be demonstrated in stained films where their presence cannot be recognised by other methods. This plan of searching for the tubercle bacillus is also of great value in the investigation of fluid obtained by aspirating the pleura and peritoneal cavity in cases of suspected tuberculosis. It is sometimes referred to as the *inoscopic* method of Jousset. The sediment obtained from the fluid mainly consists of lymphocytes in marked

contrast to the great preponderance of the polymorpho-nuclear cells constantly found in cases of cerebro-spinal fever and septic meningitis. Kernig's sign is a valuable aid in the diagnosis of meningitis, but it is not to be regarded as pathognomonic. The writer found it recently in its most typical form in some cases in which there was no evidence or suspicion of disease inside the cranium, the discovery of its presence being accidentally made in a clinical demonstration. Wall and Carr have reported several cases of meningitis in which it was absent throughout; and the former observer insists upon the necessity of excluding fallacies by placing the patient upon his back and keeping one hip fully extended whilst the other is flexed to a right-angle before attempting to extend the knee joint. If under these conditions the knee-joint can be extended beyond 135° , he regards the sign as absent. The sign is commonly present in sciatica.

Retraction of the head must also be cautiously accepted as proof of meningeal inflammation in the absence of other symptoms. It is found sometimes in caries of the upper vertebrae and often in ear-disease, and is not infrequently observed in the disordered condition associated with delayed dentition. In the investigation of this sign it is important to note that often rigidity only may be obviously present, the retraction being slight. The head in most cases may be rotated easily; but, as emphasised by Wall, difficulty in movement is only encountered when forward flexion is attempted.

Prognosis.—The disease may be practically regarded as universally fatal, though the writer believes that he witnessed one recovery. Freyhan has reported a recovery in a case in which the tubercle bacilli were found after spinal puncture; and several authorities maintain that death is not the invariable result.

Treatment.—Though much may be done for the relief of symptoms by absolute rest, the ice-cap, leeching, bromides and iodides, and the inunction of mercury when the intraventricular pressure has reached a high degree, it cannot be claimed that any drug exerts a specific action upon the disease. Ventricular drainage, trephining, and repeated lumbar punctures (as described in the author's 'Dictionary of Treatment,' on pp. 565-573) give usually but palliative results; though Oppenheim states that lumbar puncture has been followed by recovery in a number of cases.

MENINGITIS, CEREBRO-SPINAL, EPIDEMIC

Often known as Cerebro-spinal Fever, this affection, as its names imply, is an acute disease, appearing in epidemic and sporadic form, produced by the presence of a *meningococcus*, which causes a fibrino-purulent inflammation of the meninges of the brain and spinal cord. Owing to the frequency with which a petechial rash is associated with the disease in some epidemics, the affection has been often described as 'Petechial' and 'Spotted Fever.'

Etiology.—Epidemics of cerebro-spinal fever have appeared at long and irregular intervals since its first recognition in Genoa in 1805; they are usually localised, and occur in the winter or cold months of the spring. Though the disease is comparatively rare in the United Kingdom, epidemics of considerable dimensions occur upon the Continent, especially in military camps and barracks. In New York the epidemic of 1904 caused over 1,000 deaths. The recent epidemic in Belfast will be referred to later on.

Direct transmission from one person to another apparently does not take place, and it is quite rare in epidemics for two cases to occur in the same house unless at the same time. Children are much more frequently attacked than adults.

The sporadic form of the disease, according to Osler, lingers, after epidemics, for many years in some of the American cities; and since the view is generally held that posterior basic meningitis is but the sporadic form of the disease occurring in infants, it appears obvious that in its sporadic type it may be more common in England than is generally supposed.

Bacteriology.—Weichselbaum's *Diplococcus intracellularis meningitidis* is held by most authorities to be the specific meningococcus of the disease.

Netter and others have isolated the pneumococcus from the spinal fluid of patients suffering from the disease in well-marked form; hence it is still maintained by some that the infective agent in different epidemics may be one or other of these distinct organisms. A few observers reconcile the difficulty by accepting the possibility of the diplococcus being an attenuated form of the pneumococcus, though from culture-experiments both microbes appear to have quite distinctive characters. The diplococcus is found in nearly every case during the last few years when carefully looked for; often the pneumococcus has been found along with it, and sometimes even the tubercle bacillus. It is seen in great numbers in the polynuclear leucocytes obtained from the inflammatory exudation, and was till recently supposed to differ only in points of minor detail from the microbe

always found by Still in simple basic meningitis, save that it appeared to possess greater virulence.

In this connection the valuable researches of T. Houston and J. C. Rankin during the recent Belfast epidemic have illuminated many dark points in the bacteriology of cerebro-spinal fever. These observers, by utilising Sir A. E. Wright's opsonic method in the investigation of the meningococcus, obtained in numerous examples of the disease, found that the opsonic power of normal serum with reference to the meningococcus was extremely low when contrasted with its opsonic power for other micro-organisms. This property alone enabled them to differentiate the cerebro-spinal coccus from staphylococci and other cocci, since these invariably were taken up in much greater number by the phagocytes than were the meningococcus.

When the serum of cerebro-spinal patients was substituted for normal serum, phagocytosis was demonstrated to be present in a remarkable degree, the meningococci being found in very great numbers within the leucocytes; in other words, it was demonstrated that *the serum of cerebro-spinal fever patients had an exceedingly high opsonic power*. At the same time, as found by previous investigators, it was also demonstrated that marked agglutination of the cocci lying free of the leucocytes was always obvious. The opsonic power of the serum was so remarkably high that the index could only be determined with difficulty, since the leucocytes were so packed with the numbers of meningococci that counting was often impossible, though the enclosed micro-organisms were frequently not aggregated into clumps. These observers concluded 'that the opsonic and agglutinative reactions obtained in this way were to be regarded as imitations of what may be supposed to occur in the blood-stream and tissues of the patient; and the amount of phagocytosis, however it may be brought about, was to be regarded as a more or less accurate measure of the power the serum and leucocytes have of dealing with the cocci.'

It was found that the double reaction was often absent during the first five days of the disease, consequently many of the fulminant cases did not show it. Upon the other hand, as the result of the examination of seventy cases in which the disease had lasted for six days and upwards, sixty-eight gave a positive reaction. Since the published report appeared, the interesting fact has been demonstrated that two other cases giving a negative reaction have been proved not to have been examples of cerebro-spinal fever. Thus the opsonic method has been established as affording almost conclusive evidence of the diagnosis of the disease.

Houston and Rankin next attacked the problem of demonstrating whether there were different strains of meningo-

cocci. In an investigation of the blood of two patients suffering from posterior basic meningitis in the Great Ormond Street Children's Hospital, it was found that the serum did not react to the meningococcus obtained from cerebro-spinal fever-subjects suffering during the Belfast epidemic. It was also determined that the meningococci of the Great Ormond Street cases reacted with the blood of these cases, but did not react with the blood of the epidemic cases in Belfast. It was therefore considered as proven that two races at least of meningococci exist, which differ entirely in their opsonic and agglutinative reactions. (A further difference between these two meningococci has been demonstrated by other observers—viz. that though both are Gram-negative, the organism which produces true cerebro-spinal fever, unlike that which produces posterior basic meningitis, has been found by Symmers not to ferment galactose.)

Houston and Rankin next demonstrated, by procuring meningococci from subjects suffering from epidemic cerebro-spinal fever in America, Hamburg, Edinburgh, and Glasgow, that the organisms were identical with those found during the Belfast epidemic, since these invariably responded to the opsonic and agglutinative tests. They sum up at the end of their communication, which appeared in the 'British Medical Journal' of November 16, 1907, the most important points of interest in their joint contribution—viz.:

1. The opsonic method is of great value in the diagnosis of suspected cases of cerebro-spinal meningitis.

2. The method described is a most satisfactory way of testing the agglutinative reaction of cases of cerebro-spinal meningitis.

3. The opsonic power of normal serum for the meningococcus is extremely low.

4. The meningococcus may lose its power of reaction by prolonged growth on artificial media.

5. The cerebro-spinal fluid has, in all cases investigated, a much lower opsonic and agglutinative power than the blood-serum from the same case.

6. In convalescent cases the typical reaction of the serum disappears very quickly.

7. The serum of cerebro-spinal cases, when exposed to heat or kept for some time, loses its opsonic power, but retains its agglutinative power.

8. This method should prove of value in estimating the potency of any proposed therapeutic serum for this disease.

9. The true epidemic cases of cerebro-spinal fever are always due to a meningococcus having the same opsonic and agglutinative reactions.

10. The Gram-negative coccus isolated from the Great Ormond Street cases is probably the coccus of posterior basic

meningitis, and differs from the true meningococcus in its opsonic and agglutinative reactions.

A remarkable research of Professor Symmers and Dr. Wilson, made during the progress of the Belfast epidemic, resulted in the isolation by Wilson's new method of a bacillus from the water-supply of the city. This organism was at first supposed to be closely allied to the bacillus of typhoid fever, but it was soon discovered to possess the very remarkable property of agglutinating with the serum of the cerebro-spinal patients suffering during the epidemic with typical manifestations of the disease.

Morbid Anatomy.—The base of the brain is covered over with an inflammatory exudation, generally of a purulent nature. This usually extends upwards on the convolutions, whose sulci and deep fissures may be filled with lymph or bathed in thick pus, and almost invariably downwards between the membranes of the spinal cord for its entire length. The pia-arachnoid is deeply injected, and in chronic cases much thickened and the ventricles dilated, often containing pus or opaque fibrino-serous fluid. The underlying brain-surface always shows inflammatory changes, and many of the cranial nerves and spinal roots are usually also involved. In rapidly fatal cases no change may be obvious save profound congestion of the pia-arachnoid covering the brain and cord, or hæmorrhagic foci on the surface of the cerebral tissue. The lungs are often the seat of pneumonic changes in which both the pneumococcus and the diplococcus may be found, and pleurisy is common. Symmers has pointed out that marked congestive and inflammatory changes are constantly found in the intestinal mucosa, with enlargement of the mesenteric glands.

Symptoms.—Few diseases show greater variety in the nature and intensity of the clinical symptoms, so that many observers incline to the belief that often there is a double infection. As already stated, Netter maintains that in some epidemics at least the pneumococcus is responsible for the virulence of the symptoms; and this view is also held by French and Italian physicians.

The duration of the incubation-stage is unknown. In the malignant or *acute fulminant type* of the disease the onset is abrupt, with rigors or convulsions, violent headache, vomiting, muscular spasms, somnolence passing rapidly into coma, and causing death inside twenty-four hours, as in one case a few years ago seen by the writer with Dr. Parke, in a small epidemic in a country town situated near to Belfast, and in another case during the recent Belfast epidemic. A fatal issue has been recorded within five and six hours. In these cases the fever may be moderate, the pulse weak and slow, and death may occur before any eruption appears.

In the *ordinary type* of the disease the onset is also usually abrupt, convulsions being common in children, but rare in adults. Intense frontal headache, with a smart rigor and some vomiting, followed in a few hours by the appearance of marked rigidity of the muscles of the neck and back, usher in the disease in most cases of severe nature. The headache generally continues all through the attack, and is often intense even after recovery. The cervical muscles are in powerful tetanic spasm, so that the head is retracted and may almost touch the spine in the scapular region. As the lower spinal muscles become contracted the body may form an arch, but well-marked spinal opisthotonos is comparatively rare, though cervical opisthotonos is common, and is usually the last sign to disappear.

Cutaneous hyperæsthesia is a marked and characteristic feature throughout; photophobia and increased sensitiveness to noises are more marked than in tubercular meningitis. The contracted muscles are also very tender and painful, and young patients scream loudly when touched or moved. Backache and limb-pains are complained of by older patients.

The temperature is generally moderate in degree, and in some cases it may be normal for several days at the beginning and also towards the end of the attack. A continuous type of fever, as in mild typhoid, is common; but exacerbations are not infrequent, and sometimes distinctly remittent and intermittent forms of pyrexia are observed, which may resemble pyæmia and malaria. The pulse is as erratic as the temperature, often being very quick in children. Sometimes, however, it is slow; even when the thermometer is registering 106° it may only count 60. A more characteristic feature in the pulse-rate is its fluctuation, which may vary in the same patient within a few hours between 120 and 70, being at one time regular and full, and at another irregular and weak.

Marked leucocytosis is present in most cases, even at an early stage. Sighing, cyclical or Cheyne-Stokes respiration is not uncommon, but usually the respiration is little interfered with except towards the end of the attack in fatal cases.

Motor symptoms are usually present in addition to the opisthotonos; thus, as the cortex becomes irritated, epileptiform convulsions occur, and these may be unilateral. Clonic spasms may be limited to the face, or to one limb, or to both arms or both legs; they may show themselves as slight twitchings or be associated with tremors, nystagmus, or dissociated movements of the eyeballs. Strabismus is very often present. It may be intermittent, and usually should be regarded as of irritative origin, being seldom produced by definite paralysis of any particular ocular nerve. Paraplegia and hemiplegia are both very rare.

It should be remembered that even in the presence of very marked cervical opisthotonos, with the head touching the spine so that swallowing is interfered with, the patient may throughout the attack be found lying on his side, with the spinal column fully flexed. Kernig's sign is nearly always well marked.

The mental condition varies : delirium often follows marked irritability and extreme restlessness, and it may develop into furious maniacal excitement before passing into drowsiness, stupor, coma, and death. In some adult cases consciousness is not impaired ; but as the intracranial pressure increases on the supervention of hydrocephalus, cerebral failure becomes evident, and may remain permanently. Sight and hearing are often found to fail as the disease continues, though optic neuritis is very seldom met with. Both of these losses are usually gradually restored if recovery follows, unless they have been produced by inflammatory changes of a peripheral nature in the optic nerve or are the result of otitis media. Aphasia is occasionally present. Coryza is often observed, and the diplococcus has been found so frequently in the nasal secretion as to suggest this as the route by which it gains admission to the cranium, though the assumption is not corroborated by *post-mortem* evidences.

The eruption of cerebro-spinal fever has attracted much attention ; its presence and characters are as variable as the other features of the disease, and consequently afford little aid in the diagnosis. Herpes is very often present on the lips and nose, but differs in no way from that seen in pneumonia. Sometimes, however, herpes appears on the extremities in symmetrical distribution. Erythema of a diffuse type is often present about the joints and anterior surface of the trunk ; it may take the form of nodosum on the legs or urticaria on the limbs.

A general true petechial rash is very common in some epidemics, rare or entirely absent in others ; with it there may be more or less extensive purpuric blotches. In several epidemics rose-coloured papules have been occasionally observed, which, when the continuous type of the pyrexia was present, made the exclusion of typhoid fever difficult.

The vomiting present in the early stages may remain throughout the attack, but it only partially explains the great wasting that is constantly present in all save the most acute cases. Constipation is the rule, and often the urine is albuminous.

In addition to the *acute fulminant* and the *ordinary type* of the disease just described, there are a *chronic type* and an *abortive type*. Some authorities recognise a fifth variety under the title of the *mild type*. All these varieties or types of the disease were present in the recent Belfast epidemic, as reported

by Dr. Robb in his valuable paper in the 'British Medical Journal,' October 26, 1907. An example of the chronic type is given in the contribution just mentioned. The patient, a girl of eight years of age, was seized with the symptoms so constantly observed in the ordinary variety of the disease. The case dragged on till extreme emaciation terminated in death upon the eightieth day. An example of the abortive type is also detailed, in which a healthy boy of fourteen took suddenly ill with violent headache and vomiting, and became wildly delirious within two hours. Partial coma, cyanosis, rigidity of muscles, and Kernig's sign remained present for twenty hours, with falling temperature and rising pulse-rate, after which complete consciousness returned, convalescence being complete in three or four days. Dr. Robb, in his analysis of 230 cases in this epidemic, states that whatever type the disease assumed the onset was the same in all cases, with practically no variations, commencing with absolute suddenness and intense headache, followed quickly by vomiting, stiffness in the neck, back, and legs. Kernig's sign was present in all the cases with the exception of (1) those comatose and dying upon admission, (2) some very mild cases, and (3) some admitted very late in the disease. During the epidemic the writer witnessed the presence of Kernig's sign in several patients suffering from febrile ailments which proved by their subsequent course to have no connection with cerebro-spinal meningitis or other cerebral condition. Purpuric rashes were observed in less than 20 per cent. of the total cases, whilst slight enlargement of the lymphatic glands was almost constantly present. The writer several months before the advent of the epidemic had observed a series of cases of typical posterior basic meningitis in infants which led him to state his conviction that an epidemic of cerebro-spinal fever was likely soon to visit the district. In the light of the researches of Drs. Houston and Rankin, he is now inclined to look upon the occurrence of these cases as probably an instance of remarkable coincidence.

Complications are observed in a varying proportion of cases in different epidemics; the most frequent appears to be pneumonia and pleurisy, and the prevalence of the lung-complication is supposed to be greater when the infection is mixed. The influenza bacillus was found by Wall in some cases of long duration, and acute military tuberculosis was observed in association with the disease in several of his cases where the pyrexia was unusually high. Occasionally, according to Nothnagel, meningitis caused by the *typhoid bacillus* occurs during the presence of an epidemic of cerebro-spinal fever.

Joint-swelling, with pain and redness, is a marked feature in some epidemics, and the diplococcus has been detected in

the sero-purulent exudation inside the joint. In only one of the cases seen by the writer in the Belfast epidemic were joint-symptoms observed.

Hydrocephalus is often produced in the slow cases, especially in children, and it explains the high intracranial tension witnessed in many sub-acute attacks in adults. As discussed under posterior basic meningitis, the production of ventricular dilatation is supposed to be due to mechanical obstruction; but this does not explain the increase in the fluid in the subarachnoid space, over the convolutions, and in the great arachnoid and posterior arachnoid reservoirs which is usually present; nor can it account for the frequent relief obtainable by lumbar puncture, which would not take place if the foramen of Monro were blocked. Bed-sores are an almost constant complication in cases of long duration.

The course and duration of the disease are most variable: the majority of the cases which do not recover die within one week, and, as already stated, some prove fatal within twenty-four hours. As in basic meningitis of infancy, some of the hydrocephalic cases linger on for many months.

Diagnosis.—The cervical opisthotonos, intense headache, tonic spasm or rigidity of muscles, with the presence of Kernig's sign, coming on with abrupt onset and almost invariably associated with obvious spinal symptoms, cause little difficulty in the recognition of the disease in the presence of an epidemic. The sporadic cases are, as a rule, as easily diagnosed if the possibility of their occurrence be kept before the mind of the physician.

Lumbar puncture, as described under posterior basic meningitis, affords a reliable proof of the presence of the diplococcus intracellularis; and should the examination of the fresh or stained microscopic specimen be negative or doubtful a culture-experiment will clear up the difficulty. The character of the cells in the sediment of the fluid obtained by lumbar puncture is of great value in diagnosis. A large preponderance of polymorphonuclear cells is characteristic of cerebro-spinal fever and septic meningitis, while a preponderance of lymphocytes points to a tubercular or syphilitic inflammation of the membranes. Drs. Houston and Rankin have shown that the determination of the opsonic index and of the agglutinating power of the blood-serum afford most reliable diagnostic proofs of the presence of the disease, as already stated in the section dealing with the bacteriology of the affection. These methods are more valuable than the results obtained by lumbar puncture when first performed after the expiration of a week from the onset of the disease. In this way *vertical meningitis* and *otitis* are excluded. *Tubercular meningitis* occasionally closely simulates cerebro-spinal fever, and the recognition of the bacillus

in the fluid from the lumbar puncture will settle the diagnosis when this is demonstrated, otherwise inoculation of a guinea-pig will clear up the case ; but it must be remembered that a double infection is not very rare. Tubercular meningitis has usually a slow onset ; the cerebral symptoms are much better marked than the spinal, and if the latter are present they occur at a late stage, and leucocytosis is never great.

Diseases other than of meningeal origin are only likely to be regarded as cases of cerebro-spinal fever in the presence of an epidemic of this disease. More frequently the presence of the latter disease is not suspected on account of its rarity. *Typhoid fever* is closely simulated in those cases which are characterised by a continuous type of pyrexia and a long duration ; but the slow onset, the absence of Kernig's sign, the intestinal symptoms, rose-spots, and the presence of Widal's reaction, even when intense cephalalgia, vomiting, and neck-rigidity are present, settle the diagnosis. *Influenza* is differentiated by the demonstration of its own specific microbe, and *pneumonia* with cerebral symptoms betrays its presence by the characteristic lung-signs, the absence of Kernig's test, and by the result of the examination of the cerebro-spinal fluid, unless the case be one of double infection, which is a common event in some epidemics. *Primary pneumococcic meningitis* is usually of the vertical meningeal type, and may be recognised by the careful examination of the cerebro-spinal fluid.

Prognosis.—This is always serious, and in some epidemics grave from the onset ; probably always more than 50 per cent. are fatal. In the Belfast epidemic, which at the period of writing has not entirely subsided, the mortality has been very high ; the official figures up to date show that of 636 notified cases no fewer than 498 were fatal. It is therefore, upon the whole, less lethal than the other forms of meningeal inflammation, if the basic type be included as a sporadic variety of this disease. The prognosis brightens after the tenth day, unless coma and convulsions are present. In the cases of long duration the prognosis, as in the basic meningitis of infants, is that of the acquired hydrocephalus.

Treatment.—Rest in bed, the ice-cap, and other measures indicated in the management of meningeal inflammation should be resorted to ; and the ice-bag to the spine generally affords considerable relief. The diet should be fluid and liberal from the onset, as the danger from exhaustion is a serious one if the patient survive the early stage. Systematic bathing every three or four hours in water at the normal body-temperature has been found most beneficial, when carried out by a skilful nurse, in the case of children.

Leeching behind the ears or on the occiput is of value in acute cases ; but general blood-letting, except in the most

sthenic cases, is not indicated. Counter-irritation is a very doubtful procedure in view of the tendency to bed-sores.

Drugs possess little, if any, influence over the growth of the microbe, though mercurials and iodides at a later stage are most valuable in aiding the absorption of inflammatory products. Agents for the relief of pain are called for in all cases, and, as an exception to the usual contra-indication of opium in cerebral affections, this drug gives the best results. As a rule, unless the vomiting is severe, it should be combined with bromides. Morphia hypodermically is indicated when this latter symptom is persistent.

Repeated lumbar puncture is strongly advocated by Netter as a therapeutic agent, but most authorities are opposed to its systematic employment. In acute cases, with severe convulsions passing rapidly into coma, lumbar puncture undoubtedly often affords marked relief. Drainage of the spinal canal has been tried, but generally with most unsatisfactory results. Jochmann, in his report (published recently in the 'Deut. Med. Woch.'), gives an interesting account of his serum, prepared by immunising the horse by injecting living cultures of the cocci. He found the serum most useful in diagnosis, and he claims for it both prophylactic and curative powers. Three varieties of anti-meningococcus serum (those of Kolle and Wassermann, and Ruppel, and that supplied by Burroughs & Wellcome) were extensively tried in large and frequently repeated doses during the Belfast epidemic, without the slightest evidence of their therapeutic power being forthcoming. In the seventy-nine cases so treated the death-rate amounted to 74 per cent. Much more satisfactory results have been quite recently procured in the Belfast epidemic by the injection of Flexner's serum obtained from the horse, when this was injected in doses of 30 c.c. into the spinal canal after lumbar puncture. In some of the most severe cases when the serum was so employed *in the early stage of the disease* the progress of the malady seemed to be completely arrested.

MENINGITIS, SPINAL

Acute inflammation of the pia-arachnoid of the cord, or acute spinal leptomeningitis, as a primary disease, is rarely, if ever, met with.

Etiology and Pathology.—It is either a part of the general meningeal inflammation caused by the diplococcus of cerebro-spinal fever, or it may be an extension of the process in the cranium started by the bacillus of tubercle or by the pus-forming organisms which produce vertical suppurative meningitis. The writer has once seen it in a typical septic form supervene in a case of superficial carbuncle, and involve

the entire length of the cord. It may originate in fractures of the spine, after operations for spina bifida or from extensive bed-sores.

The meninges are congested and covered over, especially on their posterior aspect, with a fibrino-serous or purulent secretion, which also invades the nerve-roots, and may cause adhesions or matting of the dura mater with the pia-arachnoid. When the disease extends from the cranium, usually the exudate does not reach below the last cervical vertebra.

Symptoms.—These are overshadowed by the symptoms of the primary cerebral trouble. In the main, they are those of the spinal element which predominates in some cases of cerebro-spinal fever. There is marked pain, tenderness, and rigidity, increased by movements of the spine. Opisthotonos, cervical or general, tonic spasms of the muscles of the trunk and extremities, associated with general cutaneous anæsthesia and increase in the reflexes are usually present, accompanied by clonic seizures on the application of any stimulus. Radiating pains over the trunk and extremities, retention or incontinence of urine, and Kernig's sign may be present. Fever of a high and irregular type is common.

The meningo-myelitis which supervenes will end in a more or less general paralysis if the patient survive the acute irritative stage, which he seldom does, as death commonly results within a week from the onset.

Treatment.—This consists in the use of the agents indicated in cerebral meningitis—absolute rest, ice to the affected region, mercurial inunctions, and counter-irritation. Lumbar puncture may give marked relief.

CHRONIC SPINAL MENINGITIS (SYPHILIS OF THE SPINAL CORD)

This is usually of syphilitic nature. It may start as a chronic pachymeningitis originating in the dura mater, but much more frequently it commences as a leptomeningitis in the pia-arachnoid.

Pathological Anatomy.—This corresponds closely with that described in the chronic syphilitic meningitis, affecting usually the coverings at the base of the brain. As the symptoms produced by that affection are usually regarded as equivalent to the clinical picture of 'syphilis of the brain,' so the symptoms caused by the specific inflammation of the membranes of the cord may be taken as the clinical picture of 'syphilis of the spinal cord,' since in both cases the anatomical changes are chiefly, if not entirely, meningeal at the beginning. There are, in the main, the same combinations of pathological events as are witnessed in cerebral syphilis—viz. a diffuse new-growth, alone or, much more frequently, associated with numerous gummatous tumours and extensive arterial changes of the

obliterative type. Adhesions form between the layers of the greatly thickened meninges; the meningeal inflammation affects a varying depth of the substance of the cord, and areas of softening are not uncommon, so that the ultimate changes may be those produced by the secondary myelitis, which is sometimes intense.

Symptoms.—These may appear as early as the middle of the secondary stage of acquired syphilis; often they make their appearance before the end of the first year, and usually they occur before the termination of seven years from the date of the primary infection. Spinal, like brain syphilis is liable to appear earlier in cases which have not been vigorously treated by mercury. In most of the examples of both diseases coming under the writer's observation the primary sore appeared from the history to have been so slight as to have caused little attention on the part of the patient—a fact also noticeable in the investigation of *tabes dorsalis*.

The symptoms vary in degree and kind. Early in the affection pain is felt in the spine, radiating round the trunk, constituting the so-called 'girdle-pains,' or the pain may shoot down the extremities in neuralgic fashion, and is usually experienced in its greatest intensity at night. The injury to the nerve-roots which follows produces paresis or paralysis of the parts supplied from the levels of the cord which are most profoundly affected. Thus paralysis of one arm or one leg, or a paraplegia may result which is liable to become spastic. Sensation is usually involved, and bladder-symptoms may appear early.

The spinal disease, like the cerebral form, is characterised not so much by the special distribution of the different paralyses as by their varying features and multiform combinations and weekly oscillations. In the early stages the paralysis is incomplete, as a rule, but when the resulting myelitis is once established and atrophy or secondary degeneration has appeared, the palsy becomes complete and usually permanent. When the lesion consists of a circumscribed deposit or solitary gumma the symptoms of a spinal tumour develop. The result in this case is that the cord is slowly compressed at some localised point so that conduction is interrupted, with the supervention of paralysis of motion and sensation below the level of the growth. Sensation will be paralysed upon one side and motion upon the other if the growth be unilateral. As myelitis supervenes the paralysis becomes complete, with spasticity and increased reflexes. If the growth be situated in the upper part of the lumbar region the reflexes disappear, and there is great muscular wasting, with loss of power over the bladder and bowel sphincters.

The symptoms of the diffuse spinal meningitis vary from week to week very much more than do those caused by a

solitary gumma ; but when the resulting myelitis extends at any place deeply into the cord the symptoms in both cases show little variability, being then steadily progressive and typical of a chronic myelitis.

Diagnosis.—Where the new-growth invades chiefly the posterior root meninges and the adjoining part of the cord the symptoms closely resemble *tabes dorsalis*, with its lost tendon-reflexes, lightning-pains, paralysed bladder, ataxia, &c. ; and when the diseased process extends upwards, ocular paralysis, Argyll-Robertson pupil, and other signs may appear.

In all cases of syphilitic disease of the spinal cord, whether diffuse or circumscribed, careful investigation must be made for symptoms of cerebral mischief, which will assist in clearing up the diagnosis. The possibility of the existence of *cerebro-spinal sclerosis* must be duly considered, especially since this disease is liable in some cases to the oscillations, long remissions, and exacerbations which are so characteristic of the syphilitic disease. The intention tremor, scanning speech, partial optic atrophy, nystagmus, and other signs of this sclerosis are, however, usually distinctive.

The duration of spinal syphilis is very variable: some cases occurring in the late secondary period run an acutely fatal course ; but a slow onset and duration of years is the rule. The sudden supervention of an intense myelitis may change the clinical picture at any period in a few days, but, like brain-syphilis, the malady often responds to vigorous treatment in a way that differentiates it from nearly every other spinal organic lesion, though partial is much more frequent than complete recovery.

Treatment.—This is in every detail similar to that indicated in basilar syphilitic gummatous meningitis.

CHRONIC INTERNAL SPINAL PACHYMEINGITIS,

Or Hypertrophic Cervical Pachymeningitis, first described by Charcot, is a chronic inflammation starting in the inner layer of the cervical dura mater (rarely in the pia-arachnoid), which becomes enormously thickened by the deposit of stratified layers of a firm fibrinous exudate compressing the cord and cervical nerve-roots.

Etiology.—Oppenheim regards the condition as generally the result of syphilis, though trauma, syringomyelia, and exposure to cold may be etiological factors.

Symptoms.—Shooting neuralgic pains from the occiput to the scapula, with some hyperæsthesia or tenderness, rigidity, and muscular spasms or tremors, usher in the first stage of the disease, which may last for several weeks. The parts supplied by the median and ulnar nerves are usually most severely involved. This irritative stage is followed by paralysis of the muscles supplied by the inflamed nerves, the

skin being either hyperæsthetic or anæsthetic, and the hand more or less deformed, the wrist and metacarpo-phalangeal joints being markedly extended owing to paralysis of their flexors, whilst the middle and distal phalangeal joints are flexed, constituting the claw-hand or *main en griffe*. The affected muscles waste and show the reaction of degeneration.

At a later stage, owing to compression of the cord, paraplegia of the spastic type develops, with anæsthesia and loss of power in the sphincters. When the disease originates high up in the cervical region the musculo-spiral nerve suffers most. Occasionally the dorsal cord is the site of this hypertrophic pachymeningitis, in which case the irritative symptoms are then noticed in the intercostals, and the early motor paralysis, anæsthesia, and wasting are observed in the lower extremities and in the lower parts of the trunk.

Prognosis.—In hypertrophic spinal pachymeningitis the outlook is always grave; a few cases recover under mercurial inunctions and counter-irritation, and in these the recovery is usually incomplete.

A form of internal pachymeningeal inflammation is sometimes met with which has been described as *hæmatoma of the spinal dura mater*; it is, however, but the extension of the same hæmorrhagic process already described as occurring in the internal layer of the cerebral dura.

EXTERNAL SPINAL PACHYMENINGITIS

Etiology.—This condition is occasionally observed as the result of injury or operations upon the spinal column, as laminectomy, &c., or as the extension inwards of the sloughing process in extensive bed-sores, abscesses, and tubercular disease of the vertebræ.

Symptoms.—In the traumatic cases the disease is usually acute, and the symptoms are practically identical with those of acute spinal leptomeningitis. In spinal caries the inflammatory process is of a chronic type, but it is not rare for an acute fulminating external pachymeningitis suddenly to supervene upon a local compression-myelitis caused by tubercular disease of one or more vertebræ, as in a case recently under the writer's observation.

MESENTERIC GLAND DISEASE

This condition, which is essentially a tubercular infiltration of the lymphatic glands in the mesentery and retroperitoneal groups, is also sometimes known as *Tabes Mesenterica*, though many of the cases usually described under the latter designation are but examples of marasmus caused by enteritis, the result of improper feeding.

Etiology and Pathological Anatomy.—The disease is much more common in children than in adults. Its causation is identical with that of other tubercular lesions, but owing to the direct connection of these glands with the lacteal system of the intestine, there are obvious grounds for the belief that the disease is usually, if not always, directly produced by the ingestion of food containing the bacilli of tubercle. It is also highly probable that in the great majority of cases these are conveyed directly to the glands by the uncooked milk of tubercular animals, which forms so large a percentage of the food of children. The experiments of Sydney Martin have demonstrated that the bacilli may pass through the intestinal walls to the glands independent of any intestinal ulceration or abrasion of surface; a similar event may take place in phthisical children who swallow their expectoration.

The glands are found enlarged and caseating: occasionally only do they exhibit any tendency to suppuration; in some instances they may be found the seat of large deposits of lime-salts, as in the calcifying glands of the thorax or elsewhere. The disease in a *primary* form is often met with, as seen in those cases where the *post-mortem* examination reveals the absence of all peritoneal involvement or tubercular ulceration of the intestine. In the majority of cases, however, the enlargement of the mesenteric and retro-peritoneal lymphatic glands is seen associated with general tubercular inflammation of the peritoneum.

This latter condition may itself be but a part of a general miliary tuberculosis, the peritoneum and glands being infected through the blood-stream. It may, however, be also a secondary result of a primary mesenteric gland disease, which, originating in the bacilli carried by the lymphatics to the glands, afterwards causes a secondary infection of the peritoneum and intestinal walls. A very rapid and widespread tubercular peritonitis is sometimes the result of the rupture of a suppurating or caseous mesenteric gland.

The glands may be found enlarged and matted together, forming tumours of variable dimensions; tubercular disease in other parts of the body, especially in children, is found in a considerable percentage of cases. Behring believes that the bacilli pass freely through the intestinal tract and infect the lungs, and that some cases of phthisis in adults are examples of such infection in early life, where the disease had remained long latent.

Symptoms.—The most obvious feature is wasting, a general marasmus being present, in which the fatty and finally all the tissues, including the blood, suffer. The physical signs are generally obscured by the peritoneal complications; occasionally, in well-marked primary tabes, the enlarged glands can be palpated through the abdominal wall, but more

frequently the tympanitis and pain, or ascites which is sometimes present, prevent this. The clinical picture is therefore more frequently that of a tubercular peritonitis in which diarrhoea, with thin, offensive, brownish stools, is the prominent feature; and when there is tubercular ulceration of the intestinal mucosa the diarrhoea is accompanied by much griping pain and bloody motions, often alternating with constipation, and sometimes with obstruction of the bowel.

Fever is generally present, but, unless when there is supuration or marked peritonitis, it is usually very moderate, and the general wasting bears no proportionate relationship to it.

The duration of the disease is most variable, being influenced by the secondary peritonitis and ulceration of the bowel so often present. In the primary form the course, duration, and final issue may differ little from that witnessed in the progress of enlargement of the cervical lymphatic glands.

Diagnosis.—The chief point is the elimination of *tubercular peritonitis*, or the so-called 'consumption of the bowels,' which is so often associated with *tabes mesenterica*; as a rule, this can easily be effected by the physical signs. (See under 'Peritonitis, Tubercular.') It must, however, be remembered that large irregular-shaped omental tumours, which may be easily palpated through the abdominal wall, usually below the level of the resonance of the transverse colon, may be the result of peritoneal tuberculosis or of a widespread tuberculosis of the abdomen, without much glandular involvement.

Prognosis.—The outlook in a primary tuberculosis of the mesenteric glands is not unfavourable; many cases end in resolution and complete recovery. Even when peritonitis has supervened the prognosis is far from being a fatal one, since the results of operative and other treatments continue to show an increasing percentage of recoveries. The presence of tubercular disease elsewhere is a serious element in the prognosis, and where the abdominal glands appear to have been infected from disease in the thorax a fatal issue may be confidently prognosticated.

Treatment.—Measures indicated in other forms of tuberculosis should be employed assiduously, as open-air living, over-feeding, creosote, &c. The elevation of the opsonic index by minute doses of the new tuberculin when the index is lowered should prove beneficial. The cod-liver oil binder, in the writer's experience, is an invaluable aid to treatment. Operative methods will be referred to under 'Peritonitis, Tubercular.'

MIGRAINE. (See MEGRIM, p. 989.)

MILIARIA

This, known also as Sudamina, is an eruption consisting of very small, colourless, transparent vesicles containing sweat, which are situated in the horny layer of the skin and are produced by the temporary blocking of the sweat-ducts. The blocking occurs when the skin is dry, and the profuse sweating usually, but not invariably, associated with the eruption dilates the duct immediately below the epithelial plug which has closed its orifice. The appearance of the eruption is most characteristic. The vesicles are rarely larger than a millet-seed and are very numerous, generally on the anterior aspect of the trunk; occasionally they may be confluent. Owing to their colourless and transparent characters, they are often first detected by the touch.

Typhoid fever, acute rheumatism, and the sweating stages of pulmonary phthisis or other febrile condition, supply the most typical examples of the condition. Usually there is no inflammatory redness surrounding the globule of imprisoned sweat, and then the term 'sudamina' is to be preferred. The term 'miliaria' is sometimes reserved for the eruption when each vesicle is surrounded by some congestion.

This sweat-rash, as it is usually called, is of no importance, and may appear even when the skin is dry. It passes off usually in a few days, and is followed by an almost unperceivable desquamation; in prolonged feverish conditions successive crops may follow each exacerbation of the causative malady.

MILIARIA RUBRA

This is the dermatologist's title for the affection known as 'Prickly Heat.' It is also termed Lichen Tropicus and Miliaria Papulosa, and is identical with the so-called Red-gum, or Strophulus infantum, which affects over-clothed infants soon after birth.

Etiology.—The minute vesicles are associated with numerous sharp-pointed red papules; as in sudamina, they are produced by blocking of the sweat-orifices. They are, however, of inflammatory origin, and the contents are mixed with inflammatory exudate, which gives a milky appearance to the vesicles. These always remain discrete and do not spontaneously rupture; the accompanying inflammatory redness of the epidermis is often associated with most intense pricking and itching.

The cause of the eruption is always exposure to heat, though the parts of the body affected are usually those covered by clothing, the face and hands frequently escaping, whilst

the trunk, especially if covered by flannel, may be extensively involved.

Symptoms.—Profuse sweating invariably precedes and accompanies the appearance of the rash, which usually comes out in successive short-lived crops, associated with intense itching. It is never seen in cool weather, and is almost constantly present in Europeans residing in the tropics ; dark-skinned races are not affected.

Diagnosis.—The affection is only likely to be mistaken for the early papular or vesicular stage of *eczema*, but unless the vesicles of prickly heat are ruptured by the finger-nails in scratching there is never any weeping. The onset is sudden, and is invariably associated with the profuse sweating caused by the advent of hot weather ; its duration is transient, and the site is different from that selected by *eczema*.

Treatment.—The condition yields at once on the removal of the patient to a cool locality, when this is feasible, or on the substitution of light silk or soft thin woollen fabrics for heavy flannel underclothing.

Any soothing evaporating lotion or mild ointment will give relief, as zinc-powder suspended in elder-flower water, dilute lead-lotion, or an ointment of boric acid. The dusting over of the surface with fuller's earth, zinc-oxide, or other desiccating powder, usually is very efficacious. A smart saline purge, speedily following a tepid bran-bath, usually gives relief to the itching. Alcoholic drinks should be prohibited. A slight inunction of the body with olive oil or any bland animal fat, especially in obese perspiring subjects, often prevents further attacks whilst the weather keeps very hot.

MOLLITIES OSSIUM

This curious and rather rare affection, known also as *Osteomalacia*, consists in the presence of a softening of the bones, which leads to various deformities caused by bending and spontaneous fractures.

Etiology.—The disease is much more frequent in females than in males. It commences usually about the age of thirty, but it may start sometimes before twenty or as late as forty-five or fifty. Pregnancy acts as a predisposing or exciting cause, since the majority of instances of the disease appear during utero-gestation or soon after delivery. The many theories invented to explain its pathology are unsatisfactory ; thus it has been attributed to excess of carbonic and also of lactic acid in the blood, and nervous agencies interfering with the functions of the ovaries have been supposed to produce the disease. Nothing hitherto has been discovered about its pathology which affords a working

explanation of its comparative frequency in the districts bordering on the Rhine and in some other regions.

Pathological Anatomy.—The bones are not only softened, but they are also brittle. They cut readily with a knife, and may be indented by pinching them between the fingers like firm dough, or they may be bent like an elastic pill-mass.

The medullary canal is widened, so that in extreme cases only a thin layer of compact tissue is found lining the periosteal coating of the long bones. The flat bones of the skull may be thickened, and may be as soft in consistency as moistened leather. The lime-salts are found absent around the Haversian canals (before these disappear), as in decalcified bone, the matrix being translucent and spongy, and sometimes almost of gelatinous consistence, as observed in the scooped-out cavities seen in the bodies of the vertebræ. The enlarged spaces in the interior of the bones are filled with soft medullary matter not unlike the normal marrow. It is usually deep-red and dotted with numerous ecchymoses, whilst at places it is yellow and fatty in appearance, and the microscope reveals in it disintegrated red cells and occasionally multinucleated corpuscles. Chemical analysis proves that the inorganic constituents are reduced to 30 or 35 per cent.—*i.e.* to about half the normal proportion.

Symptoms.—The first symptoms complained of are pains of an aching rheumatic character, which fly from one bony region to another. They may begin in the vertebræ or in the limbs, rarely remaining long in one bone. The figure of the patient gradually is noticed to alter: the stature diminishes as the shoulders become rounded, or the spine becomes curved laterally and the chin is depressed on the sternum. The gait of the female patient soon markedly changes: she walks with hesitancy and difficulty, wobbling or waddling in progression; this is partly owing to the changes in the bending bones and to a general muscular weakness or progressive debility, which demands the aid of support from crutches or sticks or from the arm of an attendant.

Probably before being driven to permanent rest in bed one or several bones have snapped on slight provocation, and others have bent in various strange positions. The fractures which occur at a later stage rarely unite perfectly, the soft callus refusing to ossify, and false joints become numerous as the fractures multiply. Lime-salts in excess are found in the urine.

At a later stage the softening becomes so extreme that the tendency to fracture appears to diminish. Sometimes a long bone may be bent by the hands into a semicircle, and pressure on the facial bones may drive in their outer frail shell like the thin walls of a weak pasteboard-box. The deformity of the chest and pelvis becomes as marked as

that of the long bones, and the bent ribs and protruded sternum may break. The sacrum is driven downwards by the weight of the spinal column and the acetabula are pressed towards each other, whilst the symphysis pubis is protruded like the prow of a boat. These rostrate pelvic changes render delivery impossible except after Cæsarean section, which often ends fatally. For a considerable time there are no other symptoms, and the general nutrition is but slightly interfered with, and seldom is fever present; so that with bed-ridden patients the disease may run on a slow course for many years. Often starting in pregnancy, it may cease to progress after labour, till the next and succeeding pregnancies find the disease in its most advanced stage. It is probable that minor degrees of the condition may escape notice, especially in males.

Death is said to occur in an average of about five years, but there may be wide limits on either side of this figure.

The actual cause of death is usually asphyxia in some catarrhal attack, precipitated by the lung-compression caused by the thoracic deformity.

A condition has been met with in the fœtus in which, owing to the absence or arrest of the ordinary developmental process in the cranial and other bones, the fœtal skeleton shows remarkable deformities caused by the fractures and reunions of various bones. The condition, owing to the brittleness of the imperfectly developed osseous tissue, has been described as *Fragilitas Ossium* or *Osteogenesis Imperfecta*; it is only of pathological interest. Some of the reported cases are probably examples of congenital rickets, and others are likely to be of syphilitic origin.

Diagnosis.—A condition in which there is simple *brittleness* of the bones is not rare; it is often to be met with in senile atrophy and sometimes in chronic dementia. This is easily differentiated by the absence of softening and by the rapidity with which the fractures unite.

Malignant disease of a long bone, ending in fracture, is sometimes mistaken for osteomalacia, but a careful examination will always reveal bony enlargement.

Bradshaw has described a form of disease characterised by multiple growths originating in the red marrow of bones; these multiple myelomata may penetrate the compact tissue of the ribs and sternum and simulate mollities ossium. The diagnostic point is the presence of albumose in the urine, which has led to the title of *Myelopathic Albumosuria* for the condition.

Treatment.—No drugs are known which influence the progressive softening. When pregnancy has occurred, the narrowing of the pelvic brim may warrant the induction of premature labour. Removal of the ovaries in a few cases

has been reported as successful in arresting the disease; hence, when Cæsarean section has to be performed Porro's modification of the operation should be selected and the ovaries taken away at the same time, with the body of the uterus and the placenta *in situ*.

MOLLUSCUM CONTAGIOSUM

This is a contagious skin-disease characterised by the development of numerous small tumours, originating in the rete-mucosum layer of the epidermis.

Symptoms.—The tumours vary in size from that of a small pin's head to a large pea; occasionally they may exceed half an inch in diameter, and if two or more are situated together the resulting tumour may even reach to the dimensions of a fair-sized plum. Of firm and elastic consistency, their colour is usually whitish; and a certain degree of translucency is present, which suggested to Mr. Hutchinson their resemblance to a mother-of-pearl shirt-button. They are more or less hemispherical in shape, with a flattened or umbilicated summit, showing a minute opening, through which whitish material or a milky juice may be expressed by squeezing the growth firmly between the finger-tips, as in the case of a small sebaceous tumour. Upon their first appearance they are sessile, but as they grow a pedicle not infrequently develops. The most usual site is about the eyelids or cheeks; but the breasts, limbs, genitals, or any part of the skin may be involved, except the soles and palms, on which they are never seen.

The entire cutaneous surface may be studded over with them in rare cases. After an incubation-period which varies usually from one to two or more months succeeding exposure to contagion, the growth begins as a mere speck, requiring many weeks before its full dimensions are reached. It then remains stationary for an indefinite period; but its natural tendency is towards involution, without scarring, which, however, is usually hastened by some injury, as in scratching or friction against the clothing. Occasionally suppuration occurs after injury, or the tumour drops off from strangulation of its stalk or pedicle.

Etiology.—The contagious nature of the growths is shown by their frequent appearance in members of the same family and in school-children. The writer witnessed the outbreak of six cases in a charity-school, and Morris traced the disease amongst half a dozen of patients who had visited the same Turkish bath.

Experimental inoculation of the human subject by the contents of the tumours has produced the disease in many cases,

so that the question of contagion is placed beyond doubt, though such experiments often end in failure. The disease has also been produced by rubbing the tumour-contents into the unwounded skin of a healthy patient. Children are much more subject to the disease than adults are. Bad hygienic surroundings and absence of personal cleanliness increase the liability to contagion.

The pathology of the affection is unknown. In the contents squeezed out of the tumour certain cellular bodies have long been recognised under the name of 'molluscum bodies.' These were supposed to be protozoa, but they have been demonstrated by many observers to be but keratinised epithelial cells of the rete which have undergone hyaline degeneration. The specific parasite is therefore awaiting demonstration. The theory that the tumour was an hypertrophied sebaceous cyst is now abandoned, though its section, showing the usual orifice on the flat summit leading into a canal or duct-like tunnel communicating with the interior of the growth, renders its likeness to a racemose gland almost complete.

Treatment.—In many instances squeezing out of the juice or curdy material by pressure is all that is necessary. Splitting of larger tumours by a tenotomy-blade and curetting speedily destroy them. Any strong antiseptic, as carbolic acid, sublimate or nitrate of mercury solution, injected into the central opening causes withering of the growth. When they are very small and numerous they may be snipped off with flat scissors, or painted over with the salicylate collodion which is used for the removal of corns. They also yield speedily on exposure to the X-rays.

MOLLUSCUM FIBROSUM

This condition must not be confounded with the preceding, with which it has nothing in common; to prevent such confusion it is sometimes described as Fibroma Molluscum. It would appear that two distinct affections are described under the title Molluscum Fibrosum:

1. The non-lobulated, pendulous, isolated, and often pear-shaped tumours which are found scattered over different parts of the trunk. These may be solitary or multiple, and so small as almost to escape notice, or they may increase to the size of a cocoanut and become a source of great inconvenience. Their structure is fibrous, as implied in their nomenclature. The generally accepted view is that the majority of these growths should be classed as *Fibro-neuromata*, the diseased condition being designated as Neuro-fibromatosis, the growth starting in the perineurium of the

superficial nerve-fibres, though some of them are found to consist of nothing but fibrous tissue with a few scattered nerve-filaments. They are entirely innocent in nature; the small firm ones, which may be numerous and subcutaneous, are often associated with considerable tenderness and severe neuralgic pain. These are usually pink-coloured, and are most frequently situated on the arm, neck, face, and breast, appearing often during childhood. Removal is best effected in the pedunculated forms by elastic or silk ligature, and in the flat varieties by the knife or cautery.

2. The diffuse type of molluscum fibrosum, which has no connection with neuro-fibromatosis, must be regarded as of the nature of a simple multiple fibromatous formation. The tumours in this affection are often very large and pendulous; they may cover over a large area of the body, producing hideous deformities, as in the remarkable family first reported upon by John Murray in 1873, and again recently the subject of a most interesting record, with portraits, in the 'Medico-chirurgical Transactions' of 1903.

The bases of the tumours may be broad and most extensive, so that enormous pendulous fringes or masses of hypertrophied tissue are produced, which have been likened by Shield to the dewlaps on the neck of a bull. In one remarkable case seen by the writer the congeries of tumours in the scapular region was attached to the body by a sort of mesentery consisting of hypertrophied skin, constituting an enormous overlapping fringe-like growth of many pounds in weight which resembled the red wattles of a turkey-cock.

The loose fibrous growths are variously stated to have their origin in the subcutaneous fibrous tissue, the corium, and the follicles of the skin. In the remarkable cases recorded by Murray the growths appeared first in the gums during teething; they have remained for many years, steadily increasing on the head, face, and buttocks, and they have involved the fingers and toes, which are wrapped up in soft-lobed bluish tumours, over which there is no mobility of the skin. Other deformities sometimes are associated with the growths, as hairy moles, nævi, sebaceous tumours, and enlargement of bones; mental feebleness has been noticed in some subjects of the disease.

The smaller growths may sometimes be extirpated with scissors, the cautery, knife, or caustics; but the larger masses may in rare instances be so numerous, vascular, and extensive as to defy the courage of the boldest surgeon.

MORPHIA-HABIT

Morphinism usually originates in the use of morphia or opium which has been prescribed for the relief of physical pain caused by some functional or organic disease. The relief of the physical suffering, if the latter is continuous or frequent, calls for increasing doses, till in a comparatively short time enormous quantities are necessary for the obliteration of the pain. It is not to the condition which culminates in the capacity for swallowing or injecting these colossal doses that the terms 'Morphinism' and the 'Morphia-habit' are applicable. It is to the *craving* which the use of the drug engenders that these synonyms should be applied. Long after the disease which first called for the exhibition of the narcotic has passed away, the individual, having experienced the pleasant stimulating effects of the drug, soon finds that he cannot exist without them. As the pleasurable sensations pass off there succeed feelings of lassitude, listlessness, and depression of spirits which call loudly and irresistibly for further stimulation. The morphinomaniac or the victim of the morphia-habit then finds that he is incapable of exercising his brain or performing the ordinary duties of his calling without resorting to his narcotic ; this he uses in doses which for the time bring him up to the level of his normal mental and physical vigour.

Opium-smoking is the form in which the vice is most usually indulged in amongst the Eastern races, the habit being often deliberately started not for the relief of pain, but as tobacco-smoking is often resorted to amongst Westerns—as a luxury by the use of which ennui, mental worry, and physical fatigue, and even hunger, may be dissipated.

Without discussing the relative moral evils and degradations which accompany the various forms of morphinism, whether indulged in by smoking, opium-eating, or hypodermic injections of morphia, it must be observed that, as regards physical deterioration, there can be no doubt that opium-smoking is less injurious than the use of the hypodermic syringe. This result is obviously due to the fact that the opium-smoker rarely in an entire day consumes as much opium as would represent a single grain of morphia, a great proportion of which is entirely burned away. Allbutt, in his incomparable article on 'Morphinism' points out that the moral degradation of opium-smoking is more the result of the environment of the opium-den, which is the resort of the scum of the earth and is associated with every kind of abomination.

It is with the abuse of the hypodermic method that the physician has generally to deal, as 'opium-eating' or the

consumption of the drug in the form of tincture has of late given way to the syringe and morphia-solution.

Medical men, unfortunately, supply the largest proportion of the victims of the opium-habit, more than one-third of all morphinomaniacs being members of the medical profession who have started the use of the drug for the relief of insomnia, or often of some trivial painful affection.

Symptoms.—The morphinist is often recognised at a glance. Usually before half a year has confirmed him in his habit his bodily health has become materially affected. He is emaciated; the skin of his face is flaccid and of a peculiar yellowish-grey tint; if he originally has been thin, the skin may be drawn tightly over his facial prominences like parchment, and it is also usually of the yellowish hue of an old manuscript. He looks 'dried up' and much older than his age, and has a haggard expression; but if he also indulges inordinately in alcohol, as many morphinists do, he may exhibit the physiognomy of the drunkard. His pupils are often dilated and sometimes unequal; if examined after his usual dose they may be contracted to a pin's point.

If under observation at a time when his stimulant is overdue, he is excited, anxious, fidgety, and restless, and tremor of his hands may be observed. He is obviously unable to follow any continuous train of thought or join in a conversation of any length. A peculiar mental and physical jerkiness may betray him to the close observer. If he disappear from observation for a few moments, during which he has resorted to his narcotic, his whole being seems changed: the flush of his lost youth may have returned to his cheek, he is no longer listless and depressed, but talks with fluency, and may even appear brilliant and lead in conversation for a short period.

Digestion suffers, the appetite fails, though often he experiences thirst; constipation is the rule. As he passes from under the influence of his usual dose there is often added to the craving or hunger for the drug a painful sensation about the pit of the stomach; the writer has noticed that in women the pain is usually referred to the central zone below the umbilicus.

Sleep becomes broken, unrefreshing, and often disturbed by 'nightmare' experiences; itching of the skin often tends to aggravate the insomnia; usually every secretion of the body diminishes, but profuse sweating may be a marked exception. In women, hysterical manifestations are commonly present, especially when for any reason resort to the narcotic is temporarily interfered with; a large proportion of the women-morphinists are typical neurotics. In men impotence and in women amenorrhœa and sterility are common results of the opium-habit.

The progress of morphinism varies greatly ; in those whose wills are not entirely enslaved the dosage is kept under a few grains of morphia daily, and in such patients there may for long years be little deterioration, mental or physical, noticeable. The great majority of patients, however, reach a higher average of dosage, which may be roughly stated as about 12 to 15 grs. daily ; such cases seldom live much beyond fifty if the habit has been acquired in early middle life. The real neurotic, with less control from the start, rapidly increases the dosage till in some cases a drachm of morphia is consumed daily ; the downward course in these victims is more rapid and certain, hallucinations, maniacal symptoms, and profound debility or asthenia, hastened by anorexia and vomiting, usually terminating the miserable and unendurable existence.

Whilst the above signs and symptoms of physical deterioration are being developed demoralisation of character becomes even more obvious. The morphinist loses all idea of truthfulness and becomes an incorrigible liar, his ethical sense disappearing early. In this respect he often contrasts markedly with the confirmed alcoholic, who, though his oath cannot be believed in any matter relative to the quantity and frequency of his potations, may be nevertheless often considered as reliable in matters outside his vice. As graphically pictured by Allbutt, the morphinist soon loses all idea of time, as everyone who has any intimate relationships with him soon learns. In four or five examples of the opium-habit in physicians this was the first evidence of moral anæsthesia or demoralisation which aroused the suspicion of the writer.

Diagnosis.—The main points have already been emphasised in the foregoing brief description. A very important point is the detection of the punctures made by the hypodermic needle. In one case seen by the writer, where the habit had lasted for over thirty years, the entire surface of the body, including the face and the cutaneous surface of the penis, was dotted over with minute scars. In the marasmus-stage of morphinism multiple small abscesses are often observed to follow the needle-punctures. Delirium not unlike that witnessed after prolonged alcoholic debauches may supervene upon the suspension of the narcotic, and the hallucinations and tremors may be equally marked. Especially in women the hallucinations may partake of the melancholic type, so that mania is closely simulated, and violent hysterical out-breaks are common. The duration of this delirium is much shorter than in alcoholism, being usually only twenty-four or thirty-six hours, and is suddenly terminated by a hypodermic injection of morphia at any period. Much more serious, however, is the tendency to sudden cardiac collapse, which is a characteristic feature. In secret morphinism the

diagnosis may be only possible after a short period of strictly supervised seclusion.

Prognosis.—This is, as a rule, very unfavourable, as relapses are common after apparent cure. The duration of the habit, the extent to which the dosage has been carried, the individual character of the patient, especially as regards the degree of will-power which has survived the wreck, and the presence or absence of a family history of insanity, are obvious points of great importance, as is also the existence of any combined alcohol, chloral, or cocaine habit.

Treatment.—In the author's 'Dictionary of Treatment,' on pp. 630-635, the various methods of treating the opium-habit are discussed in detail. Seclusion in a special institution with all the necessary equipments—a resident physician, good nursing staff, &c.—is absolutely essential in every case. The great preponderance of opinion is in favour of a *rapid*, but *not sudden*, withdrawal of the drug. *Gradual* withdrawal, as advised by some, is regarded by most as a long drawn-out agony.

The plan of substituting some other narcotic is most undesirable, though occasionally the writer's method of employing large and frequently repeated doses of alcohol for a short period during the acute suffering of the total abstinence from morphia may under certain conditions be quite justifiable, but never as a routine plan. A similar method of employing large and frequently repeated doses of bromides, practised by Macleod, is less open to objection. The great danger in all cases is that of the sudden cardiac collapse which is likely to occur early in the treatment, and which must be met by prompt stimulation, either by alcohol, sparteine, caffeine, or even by a small dose of morphine. Over-feeding is essential from the start, and at a later stage the Weir-Mitchell plan is very useful. Heroin has been vaunted as a substitute and hypnosis has been extolled, but in the main it may be affirmed that there is no such royal road to be depended upon. Relapses are certain to occur should the reformed opium-eater ever be subjected to the action of the drug by a physician called in at a later period in life to relieve some painful affection. The obvious responsibility of ever prescribing opiates or other narcotics to the manifestly neurotic is so well recognised that it need not be here insisted upon. The now universal acceptance of this principle seems to be steadily diminishing the number of morphinomaniacs, except amongst the members of our own profession and amongst chemists, who are almost hourly brought into contact with the drug.

MOUNTAIN-SICKNESS

This is the name given to a group of symptoms which attacks individuals at high altitudes (after an ascent of over 16,000 feet), and which has been carefully recorded by Mr. Whymper. Gasping with open mouth from air-hunger, intense headache, a feeling of feverishness, with parching of the throat and great thirst, loss of appetite, palpitation, and, for a time, tinnitus or vertigo, are all present. After rest, languor and great lassitude continue, and though the other symptoms may partially subside they may be again brought on by the faintest exertion, which markedly increases the respiration-rate. Sometimes nausea and vomiting supervene, and occasionally hæmorrhages from the nose and ecchymosis of the conjunctiva are observed. The blood shows extraordinary increase of the red cells and marked poikilocytosis.

Allbutt, in discussing the probable factors of the different phenomena as the accumulation of fatigue-products in the blood, diminished barometric pressure, lessened supply of oxygen, indigestion, and cardiac dilatation, attaches most importance to the deprivation of oxygen, though he still thinks that the element of cardiac dilatation aggravates the condition. The symptoms usually do not pass off in the descent till a lower level is reached than that at which they were first experienced.

MUMPS

Known also as Contagious or Epidemic Parotitis, is a specific fever of mild type and highly infectious nature, characterised by inflammatory swelling of the salivary glands, with a liability to metastatic involvement of the testicles, more rarely of the mammary glands, ovaries, and other organs.

Etiology and Pathology.—The affection is most commonly seen in epidemic form affecting children and young adults. In schools it spreads rapidly, infecting nearly every child who has not suffered from the disease before. Its comparative rarity in adults may be explained by the prevalence of the disorder during the early years of life, an attack usually conferring immunity, though second and even third attacks are not very rare. Young infants are very seldom affected. The disease is obviously caused by a micro-organism which has not yet been satisfactorily isolated. Its period of incubation varies between fourteen and twenty-five days. The disease is highly infectious from its first onset, and the infectiousness may continue for more than a month after apparent convalescence.

Pathological changes in the gland have been seldom seen, as death never occurs unless from some complication at a late stage. The condition of the swollen organ is one of acute hyperæmia, rapidly passing into inflammation, with infiltration of leucocytes and serous fluid, though pus is practically never seen as the result of the inflammatory action.

Symptoms.—The first thing usually complained of is a premonitory feeling of malaise, which in less than twenty-four hours is followed by the onset of fever, headache, and sometimes vomiting, during the development of which the local signs make their appearance. These may occasionally be the first indication of the invasion, the patient waking up with some stiffness and pain below the ear on one side; the hollow over the condyle of the jaw is less marked, and there is tenderness to pressure. If the temperature be now taken it may register 100° or 102° , or even more in severe cases.

In a few hours the hollow is obliterated, the swelling extending forwards on the cheek and backwards to the sterno-mastoid muscle, so that there is a very considerable, flat, elastic, tender tumour or swelling involving the neck and side of the face, which in thirty-six to forty-eight hours has reached its maximum, by which time often the salivary glands beneath the ramus of the jaw are also enlarged and inflamed.

Usually in a day or two the opposite side begins to enlarge, so that the lower part of the face, with the upper part of the neck, is markedly widened from sterno-mastoid to sterno-mastoid, the chin merging in the general swelling, and the lobes of the ears are displaced upwards. The skin over the swollen region is usually pale, and a doughy feel is experienced on pressure, which elicits considerable tenderness. Sometimes both sides begin to swell at the same time; usually, however, there is a distinct interval between the process on each side, and occasionally this interval may be so long that the swelling on the side first affected has subsided before it begins in the opposite parotid region. The tumour, when it has involved the entire neck and lower part of the face, remains stationary for a couple of days, after which it gradually subsides, and disappears completely in about a week or ten days more. The tonsils and fauces, and occasionally the lacrymal glands, are swollen at the same time.

The pain about the jaws continues throughout, and there is great difficulty in mastication, in deglutition, and even in speaking, owing to the stiffness of the muscles attached to the inferior maxilla, which prevents the teeth being separated beyond a very limited extent, so that saliva tends to collect in the mouth, though the amount of this secretion is never markedly increased. The pain in the jaws may radiate into

the ears, but this should not be confounded with the pain of a true inflammation of the middle or internal ear, which sometimes occurs during the course of the disease, causing permanent deafness. The fever falls with the first signs of subsidence of the swelling; occasionally it is entirely absent throughout, but in rare cases it may be dangerously high, with formidable constitutional symptoms.

Convalescence is usually rapid and complete, suppuration or gangrene only occurring in extremely rare cases. The glands always resolve, the inflammatory swelling and infiltration entirely disappearing.

The writer once had the experience of witnessing nearly all the symptoms of mumps produced by placing a hypodermic tabloid of pilocarpine under the tongue. No inconvenience of any kind occurred till after the expiration of about eighteen hours, when extensive swelling suddenly set in in the parotids, sublinguals, and submaxillary glands, but chiefly in the latter, each of which in half an hour had reached the dimensions of an ordinary-sized hen's egg. The condition subsided as rapidly as it had appeared, and was not accompanied or followed by salivation.

Complications.—*Orchitis* is the most commonly recognised of these; it rarely occurs in cases happening before puberty. Appearing usually by about the end of a week after the onset, the testicle suddenly becomes tender, painful, and swollen, and not uncommonly there is some hydrocele. The orchitis was supposed to be a 'metastasis,' since it so often had been noticed to appear as the parotid tumour declined; but in rare cases it has been observed to precede the jaw-swelling, or to be the only manifestation of the infection. Resolution sets in about the fourth day, and the serious result of rapid atrophy of the testicle is frequently observed from withering of the seminal tubules. Sometimes both glands are affected, but rarely do both suffer marked atrophy. The rudimentary male mammary gland may also become inflamed.

Meningitis is a more serious, but much less frequent, complication; it is sometimes associated with the testicular inflammation. Delirium, high fever, aphasia, hemiplegia, optic neuritis followed by atrophy of the disc, facial paralysis, and permanent deafness, have been observed; but all these are rare complications.

In girls and women the mammary glands suffer, and occasionally the external genitals and the ovaries become tender and inflamed.

The writer has witnessed one well-marked case of acute pancreatitis occurring during an attack of mumps in a middle-aged thin woman, in whom the tender inflamed gland could be easily palpated; the accompanying vomiting seriously

threatened to prove fatal, but recovery ensued and resolution of the tumour followed.

Amongst sequelæ which have been observed, a general peripheral neuritis with absence of knee-jerks has been occasionally recorded. Endocarditis, which probably is a rare complication, has been reported as a sequela, and albuminuria is not very uncommon. In a few cases the delirium and mental disturbance of the meningeal complication has been found to remain, the disease passing into some form of permanent insanity or into an attack of acute mania.

During any of the above-mentioned complications which may supervene, even in the mild afebrile type of the disease, the fever and constitutional symptoms usually become marked and severe.

Diagnosis.—The affection is only likely to be mistaken for the *secondary parotid bubo*, or so-called *symptomatic parotitis*, which sometimes appears during typhoid or other severe fever or septic condition, and which may be bilateral, and generally ends in abscess. This condition is generally associated with oral sepsis, the pyogenic organisms accompanying the specific microbes which cause scarlatina, measles, small-pox, pneumonia, typhus or typhoid fever, finding an entrance to the gland through the duct opening into the mouth. Occasionally the gland becomes infected from a septic focus in the pelvis, the germs finding their way through the blood-stream.

Prognosis.—This is always most favourable, except in the rare cases where meningeal symptoms appear, and even then recovery is to be expected unless evidence of marked meningo-encephalitis develop. In a very few cases, as already stated, the cerebral symptoms have been known to eventuate in insanity. A guarded prognosis as regards the integrity of the affected testicle is necessary, since marked atrophy follows in more than half of the cases.

Treatment.—Absolute rest in bed, milk-feeding, and a mild diuretic mixture are all that is necessary in the great majority of cases. The complications occur with much greater frequency in patients who have been permitted to move about before resolution of the swelling has been completed; hence the importance of freedom from exposure to cold and of prolonged rest for at least ten days or a fortnight; though in a recent epidemic Clement Dukes found that a very high percentage of the cases developed orchitis when the most rigid precautions as regards rest were carried out. The pain and stiffness of the jaws may demand hot fomentations; usually a layer of absorbent wool covered with oiled silk or thin mackintosh is all that is necessary. Complications as they arise should be treated upon general principles. For the orchitis a suitable splint, which acts as a shelf upon which the inflamed gland can rest, is indicated. The best

local application is the *green* extract of belladonna rubbed up with six to eight times its bulk of glycerin. The B.P. liquid standardised preparations of this drug should never be employed in orchitis, owing to the danger of their being absorbed; and the same remark applies to the emplastrum, which is a much more dangerous agent than is generally recognised. Meningeal symptoms call for the ice-cap and bromides.

MUSCLES, ATROPHY OF

This includes several widely different affections, in which the pathological process may be of an inflammatory, toxic, rheumatic, or degenerative type, sometimes of primary, more frequently of secondary, origin. The most important group of diseases is that in which secondary wasting or atrophy of the voluntary muscles is the prominent feature.

Wasting of the voluntary muscles of the body, apart from that which is the result of over-use or of disuse, or of disturbance of the general metabolic function, as seen in fevers and marasmus, may be classified, according to its cause, into four definite types:

1. *Spinal* or *myelopathic*, in which the atrophic changes in the muscle are secondary to lesions in the spinal cord, which involve the ganglion-cells in the grey matter of the anterior cornua. Of this class Progressive Muscular Atrophy may be taken as the type, since its essential pathological feature is an affection of the entire motor tract, in which the spinal and bulbar centres and the pyramidal tracts are involved.

2. *Neuropathic*, in which the muscular wasting is secondary to lesions of the motor nerves, as is seen in neuritis and other peripheral nerve-diseases, and in the peroneal type of progressive muscular wasting.

3. *Arthritic*, in which the wasting is secondary to joint-inflammations which act in a reflex manner on the muscles.

4. *Idiopathic* or *primary*, in which the wasting is independent of any known lesion of brain, cord, or nerves. This form of true *myopathy* will be first described.

MUSCULAR ATROPHY, IDIOPATHIC, or MUSCULAR DYSTROPHY

Several distinct varieties or types of the disease are recognised, which have been grouped together by Erb under the title Progressive Muscular Dystrophy, and by others under the name of Progressive Primary Myopathy. Some of these affections may ultimately be found to be secondary to hitherto undiscovered nerve or spinal lesions. They are mostly rare diseases of congenital origin or affections which occur in certain families; and since in some types the atrophy is pre-

ceded by hypertrophy, the term 'Dystrophy' is more correct than 'Atrophy' when applied to the group.

In addition to their congenital or family-history peculiarity, they have the following clinical features in common: they are met with more frequently in males; are of a progressive type; sensation is not affected; fibrillary contractions or twichings are absent, as is also always the characteristic degeneration-reaction; the sphincters, larynx, and pharynx escape, and the wasting or atrophy is not confined to groups of muscles supplied by any particular spinal nerve or group of nerves. Whilst the different members of the group have the above characters in common, they are separated by symptoms, peculiarities of history, and signs which are fairly characteristic; but from a clinical point of view the distinctions are sometimes not well marked, and the rarer forms exhibit peculiarities which sometimes connect them with the commoner types. Much confusion exists by one neurologist only acknowledging a single type, of which he regards the different forms as merely varieties or phases, while another exalts these into distinct types.

1. **Pseudo-hypertrophic Paralysis.**—This form of primary muscular atrophy has better claims to be regarded as a separate type than the others, since its clinical features are more constant and its occurrence much more frequent.

Etiology.—Of its causation nothing is known; the generally accepted theory is that, like all the other so-called types, it is due to some hereditary or congenital developmental anomaly in the muscular system. It is a family disease. Males are much more frequently attacked than females—the girls in a family often escaping whilst the boys suffer, though the inheritance may as a rule be obviously traced through the mother, who herself is free. Many years ago the writer recorded a typical example of the disease in a boy whose twin-brother had escaped.

Symptoms.—The first symptoms of the disease often show themselves soon after the child has commenced to walk, but frequently they are overlooked till the age of five or six years is reached. Rare cases are recorded where the disease first showed itself between twenty and thirty; and even a later appearance has been recorded. The enlargement of the muscles may first attract attention, but as a rule the unsteady gait and frequent falls which the child experiences direct the parent's observation to the size of his leg-muscles. These are greatly enlarged, the calves being especially firm, and suggestive of increased muscular power; when the glutei, vastus internus and lumbar muscles participate in the apparent hypertrophy the lower extremities may assume the proportions of a miniature Hercules. In the upper part of the body the deltoid and triceps may be found hypertrophied;

and especially the infraspinatus; but most usually the condition, speaking generally, is one of enlargement of the muscles of the lower part of the body with atrophy of those of the upper extremities. Careful examination will usually reveal weakness and atrophy of the flexors of the hip, and of some of the extensors of the knee and hip, in the midst of the surrounding hypertrophy.

In the upper extremities the pectoralis is nearly always atrophied in its lower half with the latissimus dorsi, biceps, and serratus magnus, though the infraspinatus and deltoid may at the same time be conspicuously large. The neck-muscles and those of the face commonly escape, and it is also an important fact that, in contrast to the wasting of the muscles of the hand so constantly observed in progressive spinal atrophy, these muscles rarely, if ever, suffer in the idiopathic atrophies.

Ultimately the enlarged muscles cease to increase, and atrophy takes the place of the apparent hypertrophy in most cases.

The motor phenomena caused by the disease in the muscular system are a marked feature in the clinical picture, and it must always be borne in mind that the enlarged muscles are abnormally weak, though not usually to as great an extent as those which are wasted from the start. The child falls easily in his clumsy waddling walk, and is observed to rise with some difficulty. In ascending stairs he labours considerably, and is forced to climb by pulling upon the banisters. In standing there is marked lordosis of the lumbar vertebræ, and in advanced cases, when sitting, this may be found to become a kyphosis—*i.e.* the convexity of the curve of the spine looks backwards. One of the most characteristic points in the clinical picture is observable when the patient attempts to regain the erect position after being placed on the floor upon his back. He first turns over on his belly, and raises his trunk by the aid of his hands applied to the floor till he gets into the kneeling posture. The next step is the elevation of the knees from the ground, during which the weight of the body is supported in a stooping position by the toes and the hands resting on the floor. When in this posture for a second or two, one hand is raised and placed upon the corresponding knee, and with a jerk he manages to grasp the other knee with the corresponding hand. In this attitude, his trunk being flexed upon his thighs and his head looking towards the ground, he shifts each hand alternately higher and higher up the thighs, which he uses as a ladder, up which he climbs, till he finally reaches the erect position.

When the weakness increases in the arms he is no longer able to raise himself or stand, and often lateral spinal curvature occurs from weakness of the muscles on one side. If an

attempt be made to lift the patient off the ground by placing the hands in the axillæ, the wing-like scapulæ slide upwards to the level of the jaws, owing to weakness of the shoulder-muscles.

The calf-muscles being opposed in their action by the more feeble extensors, contraction occurs, and a position of equinus with inversion of the feet is produced which eventuates finally in partial dislocation of the ankle-joint. Contraction also occurs at the knee and elbow joints in a similar manner, causing permanent deformities.

Electrical irritability to the constant and induced current is markedly diminished as the disease progresses, but the reaction of degeneration is never observed. The knee-jerks diminish early, and eventually disappear when the extensors of the joint have become involved; the cutaneous sensation, the superficial reflexes, and the sphincters remain normal, and there is no mental weakness.

The course and duration of the malady vary much. When the symptoms appear before the fifth year, the patient by about the age of twelve often exhibits general wasting of all the muscles, the hypertrophy in those which first showed enlargement having given place by this time to atrophy. In those rare cases where the disease does not appear till the twentieth year or afterwards (the majority of these are females), the disease runs a slower course, and may even stand still for long periods. Death is usually caused by some inter-current affection, as bronchitis, and life is rarely prolonged beyond forty even in the late cases; and in the early ones death usually results before half this age is reached. Gowers states that in all cases death will most likely occur within a period of seven years after the patient ceases to be able to stand.

Morbid Anatomy. — The changes are confined to the muscles, the cord and nerves being normal. The muscular fibres hypertrophy, there is also increase in the areolar tissue, and the muscle-nuclei increase in number. As the disease progresses, the enlarged rounded fibres show marked longitudinal striation and splitting, with the formation of vacuoles, much fatty deposit and increase in the connective-tissue elements. In the same muscle the hypertrophic and atrophic changes may be found at the same time, and Erb maintains that the latter process is always preceded by some degree of enlargement, though this may in some instances be a false hypertrophy caused by fatty deposit and increased fibroid growth. In advanced examples of the disease the muscle appears to resemble portions of fatty tissue, the red colour and striation having almost disappeared.

Though, as already stated under 'Etiology,' the pathology of the condition is generally regarded as an hereditary developmental error, Erb considers that the pathological process may

be of the nature of a trophoneurosis—a theory which Batten considers probable from the condition of the muscle-spindles.

The other recognised clinical forms of Progressive Muscular Dystrophy are :

2. **The Infantile Type.**—The muscular weakness in this variety shows itself during the first few months of life ; male and female infants suffer equally, and though it is a family disease, obviously it cannot be handed down from parent to offspring. There is never hypertrophy observed in the muscles, which are weak and remain in an undeveloped condition. The main clinical feature is that the child is never able to walk, though all the muscles are capable of feebly executing the normal movements of the different parts of the body. The facial muscles are not involved, and the arm-movements are executed rather more effectually than those of the legs.

3. **The Juvenile Type of Erb.**—The disease appears as a rule at a later period than the pseudo-hypertrophic type—usually at about the age of fifteen, but it may be as late as middle-life. It is, like the other primary myopathies, a family disease ; it affects girls and boys equally. The clinical features are practically identical with those in pseudo-hypertrophic atrophy, save in distribution. There is hypertrophy, false and true, of some forearm and leg muscles, but the chief points are wasting and weakness of the upper-arm and thigh muscles. The face-muscles are not involved.

4. **Facio-scapulo-humeral Type.**—This form, also known as the Landouzy-Déjerine type, is unlike the last and first mentioned types, inasmuch as hypertrophic changes are not present at any stage. It includes two varieties of primary muscular atrophy, which may either exist separately or be found combined in the same subject—viz. (a) the type described as the 'Infantile' by Duchenne, in which the facial muscles are primarily involved. (This should not be confused with the before-mentioned Infantile type, in which the facial muscles are intact) ; (b) The still rarer form in which the atrophy affects primarily the shoulder and upper-arm muscles. In Duchenne's variety the first sign noticed is that the child does not close his eyes in sleep, but he learns to walk. As the facial muscles become atrophied and weak he presents the motionless features seen in double facial paralysis, and known as the 'myopathic' face. The muscles of the mouth are weakened, causing protrusion of the lips, or so-called 'tapir mouth.' At a later stage the shoulder-girdle muscles, the serratus magnus, rhomboids, the entire trapezius, and latissimus become atrophied, but there may be an interval between the facial and the scapulo-humeral atrophy of ten to fifteen or more years.

Like the other forms, this is a family and hereditary disease ; it may be transmitted by either the male or female parent,

and it affects both boys and girls alike. It may come on in infancy or be delayed till middle-life; usually, however, some signs appear before the fifth year, and, though usually progressive, the course is often so slow that it may interfere little with life. Cases are recorded where the affection has been known to remain stationary. The electrical irritability, sensation, knee-jerks, and superficial reflexes are in the condition described under the hypertrophic form, and the reaction of degeneration is never found—points which characterise all the types of primary muscular atrophy.

The pathological anatomy in all the types is the same, save that in some there is a previous real or false hypertrophy of the muscles; but the ultimate stage in every case reveals wasting and fatty degenerative changes in the muscular fibres without involvement of the nerves, spinal cord, or brain.

5. Hereditary Type.—This form of muscular atrophy, described of late years by Hoffmann, Werdnig, and others, may be regarded as a connecting link between the types of the *primary* disease just mentioned and the *spinal* progressive form. It is rightly described as 'Hereditary Progressive Muscular Atrophy of Spinal Origin.'

It is a family disease affecting males and females alike, and may first show itself in infancy or during the second year of life. The only symptoms are loss of power with wasting, which, unlike that in the forms just noted, is never confined to any particular muscles or groups, but is generally distributed over all the muscular system. The first symptoms of weakness are observed in some of the muscles of the back, thighs, and legs, and these rapidly spread to all the muscles of the body. The wasted muscles differ from those in the idiopathic types by usually exhibiting fibrillary twitchings and the reaction of degeneration in the advanced stage. Though sensation and the action of the sphincters are uninfluenced, the superficial reflexes may be lost. The progress is comparatively rapid, death usually following in a few years from the onset. That this is but an hereditary form of progressive spinal muscular atrophy is proved by the morbid anatomy of the affection, which shows changes in the cells of the anterior horns of the cord and bulbar nuclei, in the nerve-roots and in the nerve-trunks. Bulbar paralysis has been observed to occur. It may thus be regarded as an hereditary disease of the entire motor tract, especially involving the lower neurones, followed by *secondary* muscular degeneration.

The condition is only referred to here because of its liability to be confounded with the primary muscular dystrophies, to which it does not belong.

Diagnosis.—The chief differentiating features of primary or idiopathic muscular atrophy have already been mentioned upon page 1060 and emphasised in the brief description of

each, so that they need not be again mentioned. The family character of the entire group is a most important feature, and the elimination of the last-named form of hereditary spinal atrophy is difficult, as it also occurs as a family disease; and the same remark applies to the progressive neural atrophy of the peroneal type to be described later on. The distribution of the atrophy and electrical changes and fibrillary twitchings are, however, valuable diagnostic points in these last-mentioned affections.

Prognosis.—In primary progressive muscular atrophy this is always serious; the relative gravity has been included in the description of each: it is worst in the pseudo-hypertrophic form.

Treatment.—This is of little avail, but certainly the improvement which massage, electricity, and strychnine hypodermically can sometimes effect tends to delay the progress of the malady. Exercise of the affected muscles should be kept up as long as possible, the course of the atrophy being much accelerated after the patient takes to bed.

MUSCULAR ATROPHY, PROGRESSIVE, OF SPINAL ORIGIN

This affection, also known as Spinal Progressive Amyotrophy, or Duchenne-Aran's Disease, is caused by a chronic degenerative process involving the entire motor tract, but chiefly its lower segment, to which the muscular atrophy is secondary.

Confusion results from some phases of the disease being described as separate affections; hence the numerous synonyms in addition to those just mentioned, as Chronic Anterior Poliomyelitis, Amyotrophic Lateral Sclerosis, and Progressive Bulbar Paralysis.

Etiology.—The cause of the disease, like that of many other degenerations, is unknown; but exposure to cold, trauma, syphilis, prolonged mental anxiety and fright, have been recorded as exciting or predisposing causes. Males are affected much more frequently than females. The disease usually shows itself about the end of the third decade, seldom appearing before twenty or after fifty. Hereditary influences may be traced in a small number of cases, but the disease never presents the family characteristics constantly observed in the primary muscular dystrophies just described. There is, however, the well-marked form of this affection known as the hereditary progressive muscular atrophy of childhood, described upon page 1064, and which presents the features of a well-marked family disease.

Symptoms.—Sometimes the first symptom noticed is a dull aching rheumatic pain in the limb, which is soon to become the site of the commencement of the disease; it may be associated with numbness or coldness or other

disturbance of sensation. The main characteristics of the clinical picture, however, are weakness and wasting of the muscles. These occur together; stout patients notice the weakness, whilst spare ones often observe the atrophy, first, especially when the wasting begins in the abductor of the index-finger (the first dorsal interosseous), causing a loss of substance in the soft parts between the first two metacarpal bones on the dorsal aspect.

The muscles of the ball of the thumb are often affected first, and the wasting and paralysis steadily spread to the interossei and lumbricales. The mischief, however, almost as frequently starts in the shoulder-girdle muscles, especially in the deltoid, or it may begin simultaneously in both shoulder and hand of the same side.

The forearm-muscles usually suffer next, the extensors resisting the withering process longer than the flexors. The upper spinal muscles succumb usually after the forearms and arms, the trapezius in its lower and middle portions being generally greatly atrophied, whilst its upper part remains normal. The same remarkable phenomenon may be also observed in the deltoid and other muscles, normal portions of their tissue being found at one part, whilst other portions innervated from nerve-twigs at a different level of the cord are wasted. The disease soon attacks the opposite limb, or it may simultaneously develop in different muscular groups in each hand, forearm, arm, or shoulder, there being no regular order in the advance of the malady, or apparent law governing the involvement or escape of any muscle or group of muscles, though the platysma always remains normal or slightly hypertrophies. The facial muscles usually escape, or only suffer in the final stages of the malady. The weakness or paralysis is the result of the wasting of the muscular fibres, and usually is in proportion to the amount of atrophy. As the muscles which support the head become weakened, its tendency to fall forwards is counteracted by the patient extending the upper part of the spine so as to keep the head balanced upon it, and inclining backwards.

The abdominal muscles, intercostals, and diaphragm suffer with the other trunk-groups, so that serious interference with respiration may result. The lower extremities are much less wasted usually than the upper ones, and are involved at a later period, but occasionally the disease may begin in the extensors of the knee and leg. The muscles of the lower extremity may, however, be found often in a spastic condition with little wasting, whilst the flaccid upper extremities exhibit a marked degree of atrophy.

Deformities result from the unequal degrees of wasting in the muscular groups, permanent contractures being produced by the loss of power in the antagonising muscles. Lordosis

is common, but the most characteristic deformity is the 'claw hand,' caused by shortening of the long flexor and extensor muscles when the interossei, thumb-muscles, and lumbricales have become extensively withered. Eye symptoms are absent, and sight unimpaired; the pupils may be contracted or dilated from changes in the spinal centre.

Fibrillary twitching of the atrophied muscles is always present; it may also be the first sign that an apparently sound muscle is about to undergo wasting. The irritability of the affected muscles is increased to mechanical stimuli, a slight blow or tap causing obvious localised contractions. The electrical reactions are important, and vary according to the rate and degree of wasting. When this is very rapid and complete, the reaction of degeneration is present early; usually, however, the slowly wasting muscles steadily lose their irritability to the induced and constant current till they cease entirely to respond, but long after faradism fails to excite them the voltaic current may induce contractions. After the extinction of muscular irritability the nerves may continue excitable. Cutaneous sensibility is never diminished, though there may be pains and numb sensations complained of, and the muscles themselves may be tender to pressure.

The knee-jerks disappear early when the flaccid condition of the thigh-muscles sets in; but in the spastic condition of the lower extremities they are retained, or even exaggerated, and ankle-clonus is present. When this state of rigidity affects the upper part of the body a jaw-clonus may be obtained by tapping the chin.

The flaccid state of the wasted muscles in progressive muscular atrophy is the rule in the upper extremities. It is known as 'Atonic Atrophy.' Gowers points out that the varieties of the disease depend upon the combinations of this atonic wasting with two other types of muscular involvement. Thus there may be wasting sometimes so slight as to be unperceivable, but with muscular spasm, and a third condition of tonic atrophy with myotatic excess.

The most common combination, according to this authority, is atonic atrophy in the upper extremities with simple weakness and spasm in the legs; but every degree of atonic wasting and universal spastic paralysis without wasting may be observed affecting different parts of the muscular system simultaneously. Hence two distinct varieties of the disease have been described by Charcot, which other authorities maintain are essentially the same, the amyotrophic lateral sclerosis of Charcot being due to the involvement of the pyramidal fibres for the legs, the degeneration of the upper segment of the motor tract causing the leg-condition, whilst the change in the lower tract produces the atonic atrophy of the upper extremities.

Under 'Paralysis, Spastic,' the symptoms and signs of the condition of the muscles seen in the tonic variety chiefly affecting the lower extremities will be fully described. It is here omitted to prevent confusion ; for the same reason the description of the bulbar paralysis, which sometimes complicates progressive muscular atrophy by the extension of the spinal degenerative process upwards into the nuclei in the medulla, will be described under its own heading.

The course of the disease varies greatly ; but, as its name implies, its progressive character is always marked, though sometimes there are long remissions in its progress which raise false hopes of a recovery never to be realised. In some cases the wasting and palsy may be extreme inside a period of a couple of years from the onset ; usually one or more years intervene between the involvement of one arm and its opposite fellow. The supervention of bulbar paralysis and the weakness of the respiratory movements may at any time cause death. The sphincters are rarely involved, though sexual power is lost, and the general nutrition little influenced, save that the atrophy of the muscles may be so extreme as to give to the patient the appearance of a living skeleton.

Morbid Anatomy and Pathology.—The muscular atrophy shows the usual characteristics of degeneration in an extreme degree. The pale-yellow fibres are reduced in size, the striated markings have disappeared, the muscular substance being replaced by granular and fatty material, and in advanced cases the sarcolemma sheaths may be alone left, with a few nuclei, fatty granules, or vitreous substance in their interior, whilst here and there in the general wreck an occasional normal fibre or a group of fibres may be observed.

The anterior nerve-roots are always greatly changed, and at places no trace of their normal fibres may be visible under the microscope, the degeneration having only spared the empty sheaths. The nerve-trunks are also the seat of degeneration, which is more extensive towards the peripheries ; the posterior nerve-roots are unaffected.

The main lesion is situated in the grey matter of the cord, whose anterior cornua are always deeply degenerated, especially in the cervical region. The large ganglion-cells are found atrophied—that is, those of them which remain, as the majority usually disappear entirely. The nerve-fibrillæ are wasted, but the connective-tissue elements and neuroglia are increased. These changes may be traced downwards through the dorsal into the lumbar region, especially when the legs have been markedly involved, and upwards to the nuclei of the motor nerves situated in the medulla.

Next in importance to the degenerative changes seen in the grey anterior horns is that found almost invariably in the white antero-lateral columns. The degenerate fibres from

the anterior roots can be traced through the cornua into the anterior column. The anterior or direct pyramidal tract, as well as the lateral or crossed pyramidal tract, are always found much degenerated, and the sclerotic changes may be traced through the decussation in the medulla, the pons, crus, and internal capsule, up to the cortical motor cells.

The pathology of the affection is therefore a primary slow sclerotic degeneration, affecting the entire motor path, but chiefly its lower segment. As the degeneration begins in the lower segment and advances to the extent of causing atrophy and paralysis of the arm and hand muscles, subsequent involvement of the upper part of the path can produce no further symptoms. When the initial change has commenced in the upper segment of the tract, going to the lower extremity (either in the large cortical cells, pyramidal fibres, or in the branchings of these before reaching the ganglion-cells in the anterior horn), the legs become weak and spastic; but unless the anterior horns suffer, there is practically no wasting except from disuse. Where the wasting of the legs is very marked, but where rigidity and spasticity with increase of the knee-jerks are present (tonic atrophy), Gowers explains the condition by showing that, though many nerve-cells have disappeared, others remain normal, the upper and lower portions of the motor tract being nevertheless affected.

Diagnosis.—From the *idiopathic* or *primary muscular dystrophies* the disease is differentiated by the features mentioned upon page 1060. With the exception of the rare hereditary form of progressive muscular atrophy, the onset is usually after the age of twenty-five years. The small muscles of the hand are very early, if not the first, affected; the wasting of the ball of the thumb and little finger and of the interossei and lumbricales, the claw-shaped hand, the fibrillary twitchings and the electrical reaction all point to the spinal origin of the wasting, as do the absence of a 'family' character of the disease and of all evidence of hypertrophy of the muscles.

Cervical caries, which may lead to atrophy of the hand-muscles, is excluded by the absence of the hyperæsthesia of the spine, neuralgic pains, and other sensory disturbances, and by the presence of the local evidence of disease of the vertebræ.

Multiple neuritis is recognised by its symmetrical distribution, the discovery of the specific toxic agent, as alcohol, lead, &c., the severity of the pains, the tenderness of the nerve-trunks, and diminution in the cutaneous sensibility.

Syringomyelia is differentiated by the loss of sensibility to pain and temperature, by the trophic and vaso-motor disturbances commonly present in this disease, which, as regards the atrophy, weakness, and fibrillary twitchings, &c., closely resembles some cases of progressive muscular atrophy.

Pachymeningitis hypertrophica is differentiated when affecting the cervical part of the cord (which it commonly does) by its pain, spasms, and anæsthesia preceding the atrophy, and by the marked involvement of the parts supplied by the ulnar and median nerves.

Craft-palsies or *occupation-neuroses*, produced by over-action of the hand-muscles, are often mistaken for the beginning of progressive muscular atrophy in platers, gold-polishers, cigar-makers, &c. The atrophy in such cases is always unilateral, and is generally associated with sensory disturbances; but it is rarely, if ever, progressive, and usually disappears after prolonged rest or a change of occupation.

Prognosis.—The disease is progressive and incurable. The gravity of the case is greatly increased when bulbar paralysis declares itself, but even then sometimes the fatal issue is long delayed. Long intermissions are not infrequently met with in apparently severe cases.

Treatment.—The writer's experience corroborates the report of Gowers that the disease may be arrested for a considerable period by the hypodermic injection of strychnine in small doses, but he has not found that the arrest was permanent. Electricity, massage, and passive movements are of little use in the atrophic form, but may sometimes be beneficial in the spastic variety.

MUSCULAR ATROPHY, PROGRESSIVE, OF PERONEAL TYPE

This rare condition was first described by Charcot and Marie, and afterwards by Tooth; it is sometimes styled the Charcot-Marie-Tooth type and the Neuropathic type.

Etiology.—The disease commonly shows itself some time about the age of seven or ten years. It very rarely begins after twenty, but even later cases have been recorded.

Boys are much more frequently attacked than girls. It is a family disease, several members suffering; and it may be hereditary, being handed down generally through the male parent. In not a few cases the disease has appeared as a sequel to measles or other fever, and very occasionally it has followed influenza.

Symptoms.—The wasting appears generally first, but not always, in the extensors of the toes or the peronei and in the small muscles of the foot; not uncommonly the extensor longus pollicis is first affected. The calf-muscles after a time suffer and the thighs also waste.

The antagonistic muscles losing their normal opposition cause various deformities, as varus and equinus or equinovarus, and changes occur in the ankle-joint. After several years the atrophy appears in the intrinsic muscles of the hands: the ball of the thumb, hypothenar eminence, and interossei waste, and afterwards the forearm-muscles, but the shoulder-

girdle, neck, and face escape with the supinator longus. The claw-hand seen in progressive muscular atrophy of spinal origin results, and stress is laid upon the importance of this, with the feet-deformities, as settling the diagnosis of the peroneal type in early life. The disease is of extremely slow development; and though subject to long intermissions and sometimes to remissions, atrophic changes may continue for very many years, as both legs and both arms become gradually wasted.

The muscles show fibrillary twitchings, and the reaction of degeneration may be found in the most atrophied muscles. More usually there is only weakened irritability in the early stages, but faradism soon ceases to produce contractions. It is noteworthy that the apparently normal muscles may show electrical changes. The knee-jerks usually are lost, but they may remain when the thigh-muscles escape. Pains are common in the affected limbs, and some diminution in the cutaneous sensibility is usually present.

Pathology.—The condition was formerly regarded as of idiopathic muscular origin; but it is now accepted to be a *neuropathic* affection, most authorities considering it to be of the nature of an hereditary multiple neuritis. Beyond the profound degenerative changes in the muscles, there is evidence of degeneration of the nerves going to the affected muscles, and this degeneration has been traced backwards to the spinal posterior median columns and to the anterior cornua. Gowers believes that in some cases at least the atrophy is of idiopathic or primary muscular origin, the congenital or hereditary tendency to degeneration being called into action by the toxins of some infectious disease like measles, which thus produces the peroneal type in several members of the predisposed family. As its name indicates, the disease is progressive, but the progress is so slow that even old age may be reached.

Treatment.—This is as unsatisfactory as in the other forms of muscular atrophy already described.

MUSCULAR ATROPHY OF ARTHRITIC ORIGIN

This form of muscular wasting is a constant accompaniment of inflammatory action in a joint. It is seen in the rapid atrophy of the glutei and calf-muscles in hip and ankle disease, and of the extensors in knee-joint mischief, of the deltoid in shoulder, and of the triceps in elbow-joint affections. Seldom do any muscles suffer except those which move the joint; involvement of the entire limb-muscles does not occur. The wasting affects the muscle in its entire length; the electrical irritability is but slightly depressed, though the myotatic irritability may be much exalted. The wasting passes off as the joint-inflammation resolves.

The pathology of the condition is supposed to be explicable by the theory of a reflex influence on the muscles from the motor spinal cells, whose nutrition is disturbed by morbid stimuli proceeding from the nerves of the diseased joint.

The obvious treatment of arthritic muscular atrophy consists in such local massage and electrical stimulation as are compatible with the joint-condition. As the wasting disappears always on the joint resuming its suspended functions, little interference is usually necessary.

MUSCLES, DISEASES OF

Myositis, or inflammation attacking the voluntary muscles of the body, as a primary affection is extremely rare. Secondary myositis is more common: it is a not infrequent incident in pyæmia, ulcerative endocarditis, staphylococcic, streptococcic, and other infections.

Acute polymyositis or, as it is recently styled, *Dermatomyositis*, is an affection closely resembling in its clinical features the muscular condition met with in trichinosis.

Etiology and Pathology.—The cause is unknown. Some authorities consider that the affection is the result of a toxæmia produced by a parasitic invasion, but the nature of the toxin and the identification of the microbe have not yet been demonstrated. Tuberculosis and diabetes have been found associated with it as exciting or predisposing causes. The morbid anatomy shows that the muscular fibres are swollen and disintegrated, but, contrary to what is seen in the atrophic forms of muscular disease, there is little fatty degeneration; numerous round cells are found between the fibres, which latter often show vacuolation. There are extensive serous infiltration and proliferation of the subcutaneous tissue-elements, which explain the solid œdema seen during life; and some authorities consider that this tissue is affected first, the muscular fibres being involved secondarily.

Symptoms.—After a brief period of malaise, with a febrile temperature, pain is felt in the limbs, which are found swollen and tender. The muscles are rigid, so that movement greatly aggravates the pain, and the inflammatory œdema is hard and inelastic; it does not pit, except upon firm pressure. The skin over the affected regions shows much redness and infiltration, there being a condition of dermatitis which may be mistaken for erysipelas. Nearly all the muscles of the body may be gradually involved, and the firm œdema and dermatitis may involve the face and jaws. Sensation is but slightly if at all affected, and the knee-jerks remain. In one form, described as *hæmorrhagic*, hæmorrhage into the muscular sheaths and between the fibres occurs, causing swellings which

come and go; atrophy of the affected muscles follows; the cardiac muscular fibre suffers, and death usually results from heart-failure.

In the *non-hæmorrhagic* form the course is slower, resolution following in several weeks, though wasting of the muscles may long remain; when the diaphragm and intercostals suffer, death may result from interference with the respiratory functions.

A mild type of multiple myositis has been sometimes noted in association with attacks of severe acute urticaria; the muscular swellings disappear in a few days.

Syphilitic, gonorrhœal, and tubercular types of myositis have been described, but these must be regarded as secondary affections.

Under the title of 'Neuro-myositis' a form of rheumatic inflammation, affecting the fibrous sheaths of the fine nerves and the surrounding interstitial muscular tissue, has been recognised. One limb only is affected, and the disease runs a slow and very painful course. The ordinary type of muscular rheumatism, or myalgia, which manifests itself in lumbago, false torticollis, and pleurodynia, is sometimes regarded as a myositis of rheumatic origin. Gowers maintains that it is a true *fibrositis* involving only the interstitial fibrous tissue in which the muscle-spindles are embedded. (See under 'Lumbago.')

Myositis ossificans is a rare and progressive malady, which usually begins with the formation of indurated masses of tissue in the course of various muscles, caused by multiple attacks of myositis. These become gradually changed into bone; and as they join or become fused together remarkable deformities ensue, the arms being fastened to the thorax by the ossification and shortening of the pectorals, and the spine immovably fixed in a curved position and the head rigidly bent forward. Nearly all the skeletal muscles may become involved, but the eye, tongue, and face muscles always escape with those of the larynx, œsophagus, heart, diaphragm, and sphincters. The diaphragm may continue to carry on the respiration after the intercostals have become entirely transformed into true bone. Though progressive, the course of the ossification may halt for long periods, which are usually followed by acute exacerbations.

MYOTONIA CONGENITA, or THOMSEN'S DISEASE

This is a developmental muscular anomaly which manifests its presence by a transient rigidity (tonic spasm) of the muscles occurring on first attempting voluntary movements after a period of rest.

Etiology.—Heredity is the most important factor in the causation of the disease, as implied in its name; it is essentially

a family disease affecting several members, chiefly the boys. It was handed down through five generations of the family of Thomsen, who first accurately described it. It is not however, always congenital, as a few acquired cases have been reported, which followed severe fright or prolonged exertion of the muscles. The condition first shows itself in early childhood, sometimes before the end of the first, more frequently about the fourth or fifth, year, and later.

Symptoms.—The essential feature is the presence of a tonic spasm which occurs as soon as a group of muscles and their antagonists are brought into action after a period of rest, the will being unable for a time to cause the muscles to relax. Thus, on attempting to move from the position of rest, muscular rigidity sets in which lasts for a period of many seconds, or even for one or two minutes, rendering motion impossible. After a few attempts the spasm passes off, and does not return as long as the muscles are kept in action. The legs usually suffer most, but the hands are always more or less involved, and nearly every voluntary-muscle group in the body is affected, though the face and eye muscles usually escape. The first attempt at progression after rest may cause the patient to become rigid all over, so that he falls and is unable to rise from the floor for several minutes. If he seizes a pen or other object, for several seconds it is clutched in the rigid hand, so that all movement is arrested.

The myotonia seems sometimes to be increased by concentration of the patient's attention ; thus it is always greatest when he is conscious of being observed, and the longer the rest the more severe it is upon the next attempt at movement. It is lessened by warmth and moderate doses of alcohol, though the influence of this latter agent is denied by several observers. The muscles are found voluminous and well developed, and usually of above the average strength, though very occasionally they may be weaker than their bulk would indicate. They are more susceptible to mechanical stimuli, a smart blow causing a prolonged sluggish contraction, and the irritability to the induced current, when it is directly applied to the muscle, is increased, a weak one causing a prolonged and sluggish tonic contraction. Continual stimulation of the nerves causes the myotonic reaction of Erb: the muscles are thrown into a state of *prolonged* tonic contraction by strong induced currents, though the galvanic excitability of the nerves is somewhat diminished unless the current be a strong one ; and the nerves exhibit no increase of irritability to mechanical stimuli. Cutaneous sensibility, the superficial reflexes, and myotatic irritability are always normal.

The condition lasts permanently during life, only causing mechanical inconveniences and disabilities, which are greater if the patient does not lead an active physical existence.

Pathology.—The muscular fibres are found hypertrophied, many of them measuring double the normal width, the nuclei and interstitial fibrous elements being also somewhat increased. The nerves, brain, and cord are normal. Though similar sluggish contractions are observed in the muscles of animals poisoned by creatinin and also by veratrine, as pointed out by Hale White, there is no reason to regard the condition as caused by the retention of any waste toxic products in the body, as suggested by some authorities. Phosphate of soda has been also observed by Ringer and Sainsbury to give rise to similar sluggish contractions in the muscles. With the evidence of the above-mentioned muscular changes observed after examining portions of the tissue excised during life, the affection must at present be regarded as of the nature of some developmental anomaly in the fibres.

The only treatment of any use in alleviating the inconveniences and disabilities which the condition causes is active muscular exertion and gymnastic exercises.

Under the term *Congenital Paramyotonia*, a somewhat similar condition has been described as a family disease by Eulenberg. There was tonic spasm caused by slight exposure to cold, lasting for one or more hours, which was removed by warmth. The facial muscles were most markedly affected, the eyes being kept closed and speaking being prevented by the rigidity of the lips; the arms and legs were also affected. After the disappearance of the rigidity the muscles were found weakened for a time.

Gowers has described a condition of ataxic paramyotonia which he believes to be of spinal origin; in this, transient tonic spasms resembling those observed in Thomsen's disease were present, but they were uninfluenced by prolonged exertion.

MYOCLONIA, or PARAMYOCLONUS MULTIPLEX

Cannot be regarded as an affection of the muscles, though for convenience it is here referred to. The disease was first described by Friedreich. It exhibits itself by a series of sudden *clonic* spasms which affect the muscles of the limbs and trunk, the face usually escaping. A portion or the whole of a muscle or a muscle-group may be thrown into action, in the latter case causing movements of the limbs or entire body. The disease first shows itself about middle life, more commonly in males after severe mental emotion or fright. It is somewhat doubtful whether it should be classed as a family disease, but heredity has been traced in several examples. The short lightning-like contractions are most frequently seen in the shoulder-muscles. They may be bilateral, localised, or disseminated, and may be as numerous as a hundred per minute. Voluntary movements lessen or inhibit them, whilst they are intensified by emotional

excitement, and they do not occur during sleep. When only affecting single muscles or portions of a muscle they cause no movements, mere twitchings being discernible. In some cases tonic spasms are associated with the characteristic clonic ones. Sensation and muscular irritability to mechanical and electrical stimuli are normal. Myotatic irritability is, however, increased, the knee-jerks being usually much exaggerated.

Pathology.—The pathology of the condition is somewhat doubtful; by most authorities it is regarded as a neurosis, the ganglion-cells in the anterior horns of the cord being exalted in activity. It is supposed to be closely allied to facial spasm, and identical with Henoch's electric chorea; some regard it as more nearly allied to senile chorea. The disease is liable to be confounded with hysterical spasmodic seizures, and some authorities regard it as a form of hysteria. These divergent views are probably caused by the fact that the severity of the spasms varies so greatly in the hitherto recorded examples of the disease that a definite entity is even denied by some observers. The type of the affection which resembles electric chorea has always a grave prognosis, whilst in the minor degrees of severity recovery is not unusual under the use of the continuous current and bromides in full doses.

MYCOSIS FUNGOIDES

This affection is known by a catalogue of names, each as a rule indicative of some theory of its pathology. It is a rare disease of the skin occurring in middle-life, mostly in males, and which in its fully developed stages is characterised by the presence of numerous firm, elastic, tomato-like tumours, which develop upon patches of old inflammatory lesions of the skin.

Symptoms.—The patches at first resemble an indurated moist or dry eczema, associated with some urticaria, but after many months or some years the inflammatory action, extending to the deeper layer of the skin, causes portions of the tuberculated patches to become elevated above the cutaneous level, so that numerous raised tumours are developed. These vary in size from a small mushroom-button to the dimensions of the largest tomato; they are flattened, lobed, and may become pedunculated through shrinkage of their bases. At first covered by a shining layer of altered epidermis, their smooth reddish surfaces become excoriated, and a moist serous discharge exudes, which may lead to the formation of crusts. They may appear upon any region of the body, and may invade the visible mucous membranes. In rare cases the entire crop of growths may be noticed to have no preliminary eczematous stage, but starts from apparently healthy or

indurated skin. As a rule, however, the superficial or eczematous inflammation is for a long time well marked, and if carefully watched it may be observed to pass through what is called the lichenoid stage, in which there is much infiltration of the deeper layers when the nodular, tuberculated, or papular prominences appear, before the tumour assumes the character of a distinct elevated fungating growth.

The tumours may disappear spontaneously, so also may the eczematous or papillated patches, and new-growths may arise upon the site of those which have vanished. The disease is, however, progressive, and ultimately death results from exhaustion, hastened sometimes by pneumonia, diarrhœa, cellulitis, or other complication, but only after many fungating growths have developed.

The symptoms in the eczematous stage are itching and pain of a burning character, which may interfere with sleep ; and there is nothing incompatible with a diagnosis of a severe eczema at this period. In the fungating stage there is often no local irritation, but general enlargement of the lymphatic glands, like that observed in lymphadenoma, may occur.

Pathology.—The tumour consists of a small-celled infiltration, with scanty fibrous stroma, starting from the corium, at times simulating lymphadenoma or a granuloma produced by chronic inflammatory action ; whilst by some it has been regarded as a lymphosarcoma or round-celled sarcoma. These latter contentions are negatived, however, by the absence of fibrous proliferation and by the fact that spontaneous resolution of individual growths is constantly observed. The aspect, course, and histological changes are strongly suggestive of invasion by some parasitic organism which sets up irritative action in the corium-cells, but this has not yet been isolated, though various cocci and coccidia have been found ; probably these are of accidental origin.

The prognosis is always most grave ; treatment is generally useless save for the relief of itching and local discomforts, though phototherapy promises to be of some value in the earlier stages.

MYELITIS

This term is applied to the diffuse and disseminated inflammations which affect the spinal cord, and does not include conditions like poliomyelitis, which select certain definite tracts or systems in the spinal centre.

Etiology.—No age is exempt, but the disease is comparatively rare in childhood. Cold, over-exertion, and prolonged sexual excesses probably only act as predisposing causes. Trauma as a causative factor is seen after direct injury to the spinal column causing fracture. Disease of the vertebrae,

especially of tubercular nature, and tumours, especially if of a malignant form, originating in the spinal column or in the cord itself, frequently produce myelitis. Meningitis produced by the extension of any disease from outside the spinal canal, as posterior mediastinitis and bed-sores, may set up a diffuse inflammation of the cord. Syphilis is an undoubted cause, and the cord-implication, which is also usually accompanied by meningitis, may appear even as early as the later stage of the secondary period, as described on page 1038.

All the above-named causes are, however, of less importance than the next etiological factor. The great majority of typical cases are found as the sequel to the influence of the micro-organisms which cause the acute infectious fevers, and influenza, diphtheria, gonorrhœa, malaria, &c. Whether the inflammation is caused by the microbes or their toxins is a difficult problem; the spinal symptoms, however, do not generally manifest themselves till after the lapse of a period when the bacteria are supposed to have left the body. Exposure to the vapour of certain substances like chloroform, nitrobenzene, and intoxication by coal or sewer gas and sulphuretted hydrogen, have caused myelitis.

The disease may be acute, sub-acute, or chronic, disseminated, diffuse, or focal.

Symptoms.—These vary with the part of the cord affected, the most common lesion being an *acute transverse myelitis* involving the cord in the *dorsal* or *thoracic region*. Some preliminary numbness, tingling or slight pain, and twitchings may be felt in the legs for a brief period; but, as a rule, the onset is abrupt, with anæsthesia and weakness in both legs, which rapidly develop into paralysis. In very acute cases following an infectious fever, there may be a chill, with moderately high temperature.

The anæsthesia may be complete within twenty-four hours or less, and often at the level of its upper border some hyperæsthesia or girdle-pain may be present. On passing a hot sponge down the spine, when the hyperæsthetic zone is reached the patient experiences marked pain.

The paralysis is usually complete, and a spastic state of the muscles is present, with great increase of the reflexes, though these may be temporarily lost from inhibition within the first few hours. When the transverse myelitis extends through the entire thickness of the dorsal cord the reflexes may be lost completely, and may remain absent throughout; this complete loss is, however, a rare condition, and may practically be regarded as evidence of involvement of the lumbar region.

There is little or no wasting of the muscles, their electrical excitability is almost normal, and no reaction of degeneration is present—a condition in marked contrast to that observed

when the grey matter in the lumbar enlargement is involved. The case presents the typical features of an ordinary paraplegia, with total loss of power in all the muscles below the cord-lesion; and at a later stage the clinical picture is that of spastic paraplegia, with spasms, &c.

The sphincters are involved early; retention of urine at first is followed by incontinence of urine and fæces, both of which may be voided without the patient's knowledge. Cystitis is common, impotence occurs, and occasionally priapism; bed-sores are liable to form from the slightest pressure. Trophic changes, vaso-motor and secretory disturbances, joint-mischief, and, at a later stage, contractures, may be present. The temperature of the skin over the legs may be raised at first; later it becomes sub-normal.

Transverse myelitis in the *lumbar region* causes a flaccid paralysis, with rapid wasting of the muscles, which exhibit the reaction of degeneration. The superficial and deep reflexes are lost, the anæsthesia is confined to the legs and lower part of the abdomen, the implication of the sphincters is rapid and profound; cystitis with ammoniacal urine, and ulceration of the bladder, are present, and bed-sores are the rule. There is no girdle-pain, but neuralgic pains may be complained of, darting down the legs before sensation is abolished in the skin.

When the *conus* only is involved there is loss of power in the sphincters, impotence, and absence of the Achilles tendon-reflex, and loss of sensation in the parts supplied by the third, fourth, and fifth sacral nerves, whilst the lower extremities are intact. The cremasteric reflex is present, though the skin of the scrotum is anæsthetic.

Transverse cervical myelitis in the region of the last two neck-vertebræ causes paralysis of both arms and legs; when extensive, the arm-paralysis is characterised by wasting and flaccidity, whilst the legs are spastic. Anæsthesia exists everywhere below the lesion—viz. in the arms, trunk, and legs. The intercostals and abdominal muscles being paralysed the breathing is entirely diaphragmatic, and the pupils may be contracted.

If the mischief be situated in the upper part of the cervical region, should the patient survive, there is no wasting of the arms, which soon become spastic like the legs. The diaphragm is also paralysed, and all power of respiration is abolished should the paralysis be complete. The medulla, with its vital centres, will be involved if the myelitis extend upward; vomiting, tachycardia or bradycardia, difficulty in swallowing and in speaking, hiccough, and high temperature may be noticed.

Great variations in the first symptoms may be observed if the myelitis travel either upwards or downwards and if

its extension be mainly in the direction of the grey matter in the anterior horns or in the posterior region of the cord. If the lesion be incomplete or limited to one side of the cord, corresponding degrees of mildness in the symptoms may be observed, paresis taking the place of complete paralysis, and dulling of the cutaneous sensibility only being observed instead of anæsthesia.

Focal myelitis, where the inflammatory action is confined to a single spot, may produce very slight symptoms, and these may be entirely confined to the poliomyelitis anterior acuta type.

Disseminated myelitis, where numerous foci are distributed throughout the different regions of the cord, occurs after the infectious fevers, and may show itself by simultaneous impairment of the functions of the cord, medulla, and even cerebrum; more frequently the different regions of the cord are affected in irregular succession. Oppenheim points out that these cases often closely resemble acute multiple sclerosis, and may be identical clinically with the *acute ataxia* which sometimes follows peripheral neuritis; he has seen this type of myelitis change into multiple sclerosis. Optic neuritis is sometimes present.

Diffuse central myelitis runs an acute course with high fever, total paralysis of muscular power, sensation, and reflex action appearing sometimes simultaneously in the arms, legs, and trunk.

The duration, course, and ending of the various forms of acute myelitis vary quite as much as their symptoms. Death may occur in a few days from failure of respiratory power; more frequently in the fatal cases the issue is delayed till bed-sores form and terminate life by pyæmia or exhaustion. Death in many instances is due to the bladder-inflammation, which is very liable to involve the ureters and kidneys.

Recovery may follow where the above complications do not occur, but it is rarely complete, except in the mild cases. The motor paralysis may remain permanently complete: usually sensation returns early in a more or less marked degree. The most usual result is the gradual development of extensor spasms in the legs from secondary degeneration of the pyramidal fibres, constituting typical spastic paraplegia, which may ultimately end in permanent contractures from the supervention of flexor-spasms. These contractures are indicative of complete and permanent injury to the cord, from which recovery is impossible.

When recovery is apparently progressing satisfactorily in mild cases, or has become complete, recurrence of the myelitis is not a rare event. In the acute disseminated form, involving the entire nerve-centres, brain, medulla, and cord, occurring in children after fevers, especially after measles, in which

there may be delirium, ataxia, intention-tremors, scanning speech, or aphasia, complete recovery has been observed; but it is rare, though death is also unusual. In the myelitis following gonorrhœa recovery is the rule. Syphilitic and influenzal forms often completely recover under careful treatment, and, upon the whole, the prognosis is better in acute transverse myelitis which follows an infectious fever than in the insidious varieties, which are more prone to relapses. The writer has witnessed complete recovery in an adult where the lower cervical cord was affected, causing diaphragmatic breathing for several weeks, and where the limb-paralysis had lasted for more than a year. The cause of the disease in this case was attributed to violent exertion followed afterwards by a chill.

Chronic Myelitis is sometimes a sequel to the acute affection; but the recognition of a special type of myelitis by this name has led to confusion, as some writers still include under it the sclerotic degenerations and the conditions of the cord characterised by increase of its interstitial tissue.

As regards causation, gout, alcohol, injury, exposure to cold, syphilis, and lead-poisoning have been held to be etiological features in those cases of myelitis whose tendency has been to run a very slow and chronic course; but the nature, seat, and distribution of the lesions in the cord, and their symptoms, in the main correspond with those found in the acute disease. Most of the cases formerly described as examples of chronic myelitis are now regarded as instances of cerebro-spinal sclerosis, in which the cord-symptoms overshadow the cerebral signs.

Morbid Anatomy and Pathology.—The affected portions of the cord are somewhat swollen and to the touch distinctly softened, so that after the removal of the membranes they may appear almost of liquid consistence. The grey and white matter cannot be differentiated from each other, being usually yellowish-red from hæmorrhagic infiltration of a capillary nature. Where the inflammatory process is less intense it may be obvious that the grey matter shows most change, being at places distinctly hæmorrhagic. The white matter is, as a rule, changed most in those cases where the disease has commenced in the membranes or in the vertebral bodies. Under the microscope there are seen swelling and disintegration of the axis-cylinders, which show fusiform enlargements. Proliferation of the blood-vessels, whose sheaths are surrounded by accumulations of leucocytes, and numerous granular cells are everywhere visible. If the ganglion-cells can be recognised, their nuclei may be seen in a state of multiplication, and in the midst of their vacuolated and granular protoplasm numerous fatty globules are seen. If the disease has been of long standing, degenerative changes

may be observed ; the colour of the affected cord is changed from red to yellow or to white, and there is marked proliferation of the neuroglia, simulating sclerosis. In the diffused form the grey matter may be found altered in the different myelitic foci scattered throughout the lumbar, dorsal, and cervical regions. The nerve-roots suffer, and in long-standing cases ascending *degeneration* may be traced into the posterior columns and descending changes into the pyramidal tracts ; but the *inflammatory* process may advance in an opposite direction for short distances.

These changes show the pathology of the disease to be an acute inflammation, probably the result of morbid blood-conditions produced by microbic action. The micro-organisms themselves are rarely detected, though streptococci and staphylococci are not infrequently observed. This led Grasset to conclude that the myelitis is probably the result of a mixed or secondary infection.

Diagnosis.—*Spinal Meningitis* is differentiated by the severity of its sensory manifestations, severe neuralgic pain being always present, radiating in the direction of the nerve-trunks connected with the irritated roots, and there are muscular spasms, with rigidity. *Hæmorrhage* into the substance of the cord produces sudden severe pain, followed in a few minutes by paralysis, the onset being much more abrupt than that seen in the most acute instances of myelitis which have been sometimes described as hæmorrhagic. *Landry's Paralysis* may be excluded by the presence of marked impairment of sensation, which is but slightly marked or quite normal in that disease, as are also paralysis of the sphincters and the tendency to bed-sores ; the electrical reactions of the muscles are normal, and there is no wasting, unless the patient has survived for a considerable period.

Multiple Neuritis is differentiated by the symmetrical distribution of its paralysis, the intensity of the pains, and tenderness of the nerve-trunks and often of the muscles. The increase in myotatic irritability, with spasms in the leg-muscles, even when there is also paralysis of both arms, shows that the mischief is of spinal origin. In multiple neuritis the sphincters are nearly always intact ; the anæsthesia is seldom complete, there are no trophic changes, the wasting of muscles is not so acute, and there is the history of a chronic toxæmia.

Hysterical Paraplegia does not exhibit any tendency to trophic changes or bed-sores ; there may be retention, but not incontinence, of urine or of fæces, and girdle-pain is absent. There is never the characteristic extensor-spasm of the legs which may be seen in myelitis. Gowers lays stress upon this latter differential point, as tested in the following manner—*i.e.* if one leg of the patient be lifted from the bed

and the other moves with it owing to its rigid extensor-spasm, hysterical contracture may be safely excluded. Increased myotatic irritability, exaggerated knee-jerks, and ankle-clonus are also of great value in the exclusion, but not so important as the presence of severe extensor-spasm.

Disease of the vertebræ causing compression-myelitis will be referred to in the following article.

The localisation of the myelitis in the transverse variety is easily accomplished by remembering the symptoms described as characteristic of the dorsal, lumbar, and cervical types, already described.

Prognosis.—The chief points have been emphasised in the description of the progress of each variety, and need not be repeated. As a rule, the greatest immediate danger to life is in the paralysis of respiration, and this, of course, is intensified when the cervical cord is involved, so as to injure the phrenic origin; medullary symptoms are also most grave. The completeness and duration of the anæsthesia and paralysis are important; when these last beyond a few weeks without any improvement being noticeable the recovery is not likely to be complete. Compression-myelitis warrants usually a much brighter prognosis.

Treatment.—The patient should be put upon a water-bed and kept in a position of absolute rest, his body being gently turned from time to time to prevent the constant pressure which is so liable to eventuate in bed-sores. Absolute sterilisation of the catheter is most vital, owing to the tendency to bladder-disease. In acute cases ice or warm applications to the spine, leeching, and cupping have their advocates. In chronic cases counter-irritation by blisters and cauterly, and in the stage of resolution galvanism to the spine and faradism and massage to the muscles, may be tried. Drugs are of little use, though occasionally marked benefit may be obtained from mercury and iodides, even in cases where there is no suspicion of syphilis. Strychnine injected into the wasted muscles is undoubtedly of value in the stage of convalescence. The treatment of bed-sores, cystitis, &c., is to be conducted upon general principles, but good nursing and the most scrupulous cleanliness are of more importance than medication in all cases of myelitis.

MYELITIS CAUSED BY COMPRESSION

Dislocations or injuries of the first two cervical vertebræ usually cause sudden death by *rapid* compression of the cord. Fractures just below these—*i.e.* of the third or fourth—cause death from injury to the phrenic nerve. Fractures in other regions cause the loss of conduction, with paralysis of

sensation and of all muscles whose motor-nerve supply arises from the segment below the injury. The symptoms in each case correspond mainly with those observed in complete transverse myelitis; but the paralysis is usually a flaccid one, with loss of all the deep and most of the superficial reflexes owing to the completeness or severity of the cord-injury destroying all conduction.

Slow compression of the spinal cord is the result of the pressure of new-growths, generally of a malignant nature, aneurisms, hydatids, or hypertrophic meningitis. By far the most common cause, however, is tubercular disease of the bodies of the vertebræ, ending in caries and constituting

TUBERCULAR SPONDYLITIS

This may be taken as the type of a compression-myelitis.

Etiology.—The etiological factors present in all forms of tubercular infection are operative in this localised tuberculosis, and need not be enumerated here. One frequent cause is trauma, as a large percentage of cases of spinal caries supply the history of a direct injury, as of a blow on the spine or of some violent twist or strain. The diminished vitality of the injured part permits of the multiplication of the bacilli which are accidentally finding their way into every individual through air, water, and food; in this respect tubercular spine resembles the tubercular testicle which so often follows direct injury. No age is exempt, but it is more common in early adult life.

Symptoms.—The evidence of disease of the vertebræ is usually complete in the later stages, when the angular deformity or kyphosis shows itself; this was first described by Pott and has since been recognised as 'Pott's curvature.' The signs and symptoms of myelitis may precede the deformity; more frequently they appear afterwards. The first symptoms are usually those which are the direct result of the bone-disease, and chief of these is pain, which is greatly aggravated by bending the spine or by pressing upon one or more spinous processes. Localised tenderness may be elicited in this manner before the patient complains of any pain. The presence of severe agonising pain of a neuralgic character is much more frequently observed in malignant disease of the spine and in eroding aneurisms. The pain is caused by irritation of the nerve-roots, and is usually accompanied by hyperæsthesia, and often by anæsthesia. Various spots may be the seat of intense pain, though the sensibility to touch and pricking may be absent; these constitute the areas of anæsthesia dolorosa. Herpes zoster and trophic changes may be observed, and wasting of muscles occurs.

As the caries proceeds and the bodies of one or more vertebræ are destroyed the angular deformity becomes obvious; but it must be always remembered that the spinal symptoms are

very rarely produced by the mechanical compression thus brought about. They are the result of a slow compression by the inflammatory products which form between the bodies of the vertebræ and the spinal dura, and a distinct myelitis is set up thereby. Hence, in the main, the symptoms are those of a narrow zone of transverse myelitis, which ends in softening of the cord at the level of the caries of the bones. The symptoms, as already described under 'Myelitis,' will, of course, vary according to the level of the cord affected; and as this is most commonly in the dorsal region, there will be a slowly developing paralysis of the legs, with spasticity or rigidity and increased reflexes. The sensory disturbances consist of tingling, numbness, or incomplete anæsthesia of the zone supplied by the nerves of the affected segment of the cord, with hyperæsthesia at the level of its upper border, and severe pain in the areas of the distribution of the intercostal nerves as their roots become irritated; a girdle-sensation is common, and there is implication of bladder and rectum.

When the last two dorsal or first lumbar are carious the leg-muscles are wasted and flaccid, the reflexes are diminished or abolished, the sphincters are seriously impaired, trophic changes and bed-sores are common, and the anæsthesia may be complete.

In lower cervical caries there is atrophic paralysis of the arms, with spastic weakness of the legs, contraction of the pupils, and vaso-motor signs, with also involvement of the thoracic and abdominal muscles.

When the cervical cord is involved above this, both legs and arms become spastic, but sometimes the legs may long remain free, and in rare cases they may entirely escape; when the phrenic is involved the breathing becomes seriously embarrassed. In disease of the first and second cervical the neck-muscles are in a state of spasm, fixing the head immovably; there is severe occipital neuralgic pain when the nerve-roots become irritated, though anæsthesia may occur later. When the spasm is absent crepitus may be felt on movement of the head. Cough, dysphagia, interference with cardiac action, and symptoms of bulbar paralysis may supervene and cause sudden death.

In cervical caries a retropharyngeal abscess may form; whilst in the involvement of the lower parts of the spinal column psoas or other abscesses may point below Poupert's ligament. Deformity is much less marked in cervical than in dorsal cases, but great thickening may be detected over the diseased vertebræ.

Rarely is the conduction entirely destroyed; hence the symptoms are usually those indicative of a partial lesion. Sometimes they are unilateral, the pain and paralysis being

confined to one side, whilst the opposite and corresponding region is the seat of anæsthesia. In mild cases of spinal caries motion is as a rule always more deeply involved than sensation, and often the patient only complains of some stiffness of the lower limbs as long as he remains in bed; but on attempting to walk evident weakness of the muscles shows itself along with the spasticity. Fever is sometimes entirely absent; more frequently, however, it appears at some stage of the disease along with the other constitutional disturbances usually associated with tubercular infection, in which case lung or visceral implication may be suspected.

The course of the disease is usually a chronic one, extending over very many months or a few years; complete recovery is common, though the deformity of the spinal column is usually permanent. Resolution of the bone-mischief can, however, be complete, though more or less paralysis may permanently remain. Liability to relapses should not be forgotten; the writer has seen a fatal myelitis, with suppuration of a vertebral body, supervene upon an old bony ankylosis and perfectly restored spinal cord of thirty years before.

Diagnosis.—There never can be a difficulty, unless in the absence of the characteristic deformity; and long before its appearance the painful nerve-root symptoms, local tenderness, fixed and rigid position of the spine, the spastic condition of the paralysed limbs, and the cord-manifestations, settle the diagnosis.

Malignant tumours and aneurisms affecting the cord and spine cause a transverse compression-myelitis which may closely simulate tubercular caries, but as a rule the pain is much more intense, and the symptoms are more likely to be unilateral. In this latter case the motor paralysis is on the side of the lesion; the muscles innervated from the injured portion of the cord waste and become flaccid owing to destruction of their ganglion-cells, but the muscles supplied by the lower level of the cord, owing to degeneration of the pyramidal tract, become spastic. Anæsthesia in a band of skin corresponding to the affected level of the injured cord is observed *on the paralysed side*, whilst below this band the sensation is exalted or normal, save that the muscular sense is impaired; the reflexes are exaggerated and the skin-temperature raised.

On the opposite side of the body there is complete loss of painful and temperature sensations, and some tactile insensibility in the portions of the body below the level of the lesion, though the muscular sense, reflex action, and temperature of the skin are normal. This symptom-complex is usually known as Brown-Séquard's paralysis; it is sometimes, but rarely, observed in spinal caries, whilst in malignant disease,

tumours, syringomyelia, and bayonet or punctured wounds of the cord it is more common.

The youth of the patient and the presence of tubercular disease elsewhere is strongly suggestive of spinal caries, whilst malignant disease of the breast or other organ practically settles the diagnosis of cancer before the deformity appears, for kyphosis may also be the result of malignant disease of the spine.

Involvement of the cauda equina and of the conus, which occupy the spinal canal from the level of the second lumbar to the coccyx, is rare in spinal caries; the result of impairment of the conus is described upon page 1079. The cauda is not infrequently involved in tumours and fractures, and occasionally in extensions of the tubercular processes low down in the spine. The symptoms vary with the level of the lesion. If at the level of the second lumbar, the legs are paralysed and wasted, the sphincters are involved, and anæsthesia may extend almost to Poupart's ligament. When the lesion is below the third lumbar vertebra the paralysis is partial, the buttocks, thigh-flexors, and feet-muscles suffering with the sphincters. Cauda lesions exhibit motor paralysis as a rule more obviously than sensory impairment, though there may be severe pain present.

Prognosis.—Though the outlook is serious, it may be said to be better than in other organic affections of the spinal cord associated with marked paralysis. It is best in young subjects and when the dorsal cord is involved, but cervical caries, when the diaphragm is paralysed, or the first and second vertebræ implicated, is a very grave lesion. In the writer's experience the prognosis is always better in the patients who take to bed early. Trophic changes, as deep and extensive bed-sores and cystitis, are serious complications. Half of the cases in young subjects probably end in complete recovery, whilst but a small percentage of aged patients survive. Noble Smith stated that he never had a case of paraplegia from spinal caries which did not recover; and he says spinal caries, in every case where the patient's health is not absolutely undermined, is curable without increase in the deformity. Compression-mylitis caused by malignant disease and aneurisms is always fatal.

Treatment.—The constitutional treatment is that indicated in all forms of tuberculosis, and need not be here detailed. Absolute rest in bed is essential; if this for any reason is impossible, prolonged fixation of the spine must be effected by suitable appliances. In the majority of cases both aids are absolutely necessary. Thus, splints or the plaster jacket should be worn day and night. The details of the method of adapting a suitable spinal support are discussed in the author's 'Dictionary of Treatment,' pp. 130-134. The jury-mast

apparatus for caries is very suitable when the lower cervical vertebræ are diseased. Laminectomy has been resorted to with very satisfactory results in suitable and carefully selected cases, which, however, only constitute a small percentage of all cases of spinal caries. The method of forcible extension of the spine, so as to remove the deformity, is attended with considerable danger, and is seldom indicated. Careful nursing, good feeding, and the use of a water-bed are agents productive of better results than those obtainable by surgery.

MYOCARDITIS.

(See under HEART DEGENERATIONS, p. 552.)

MYOSITIS. (See under MUSCLES, DISEASES OF, p. 1072.)

MYOTONIA. (See under MUSCLES, DISEASES OF, p. 1073.)

MYXŒDEMA

This is a diseased condition caused by absence of the internal secretion of the thyroid gland, and characterised by symptoms resembling those observed in cretinism. The history of myxœdema furnishes one of the most interesting chapters in modern medicine. First described by Gull, and afterwards studied by Ord, step by step its pathology has been cleared up, as the experiments of Kocher, Horsley, and others demonstrated the function of the thyroid by noting the series of changes which occurred after its entire removal in man and animals.

Etiology.—The disease is much more common in women than in men (nearly 10 to 1). Rare before twenty, its most frequent advent is between thirty-five and fifty. Heredity plays a minor part, but sometimes it appears to approach in its etiological features to the characters of a family disease, one member suffering from the disease whilst others may suffer from goitre. (The congenital form of myxœdema is known as cretinism, and is described upon p. 251.)

Child-bearing probably acts as a predisposing cause, since it is more common in married women. In some instances it follows recovery from exophthalmic goitre. In others it may follow excision of goitres, when the entire gland has been removed, in which case it has been described as a separate condition, under the title of Operative Myxœdema or Cachexia Strumipriva. This condition, however, only supervenes in

about 17 per cent. of all thyroid operations, and probably never occurs when a portion of the thyroid or one of the parathyroids has been left. The disease is more common in Great Britain and Ireland than upon the Continent, and is very rare in the tropics.

Symptoms.—The clinical picture, as first accurately described by Ord, is recognised by the characteristic dull, expressionless, coarse physiognomy and yellowish-white colour of the skin. The nose is broadened, the lips thickened, and the eyelids swollen; a pink flush is seen over the malar prominences, the ears stand out prominently from the side of the head, and the eyebrows are arched and elevated. Often the eyelids are oblique, and the hair becomes dry, brittle, and gradually baldness supervenes; the teeth decay or fall out. The facial outline is suggestive of renal œdema, but the swelling is solid and does not pit even on firm and prolonged pressure. The entire skin of the body is also thickened or swollen in a similar manner, and does not pit; there is great increase in the subcutaneous fat, which is collected, often in large patches or plaques, above the clavicles. This inelastic swelling of the skin gives an appearance of coarseness and clumsiness to the entire body; it is best seen in the hands which become spade-like, and in the feet which enlarge and broaden out in a similar manner. There is no perspiration, the skin being dry, rough, and often scaly. Tactile sensibility is delayed, all movements are slowly and clumsily executed, and the special senses are more or less impaired.

With these obvious external changes mental symptoms develop. The patient exhibits a remarkable slowness of thought and a highly characteristic deliberation in speaking, which latter is also somewhat intensified by the swollen condition of the tongue and lips, and the thickening of the mucous membrane of the mouth, palate, and throat. Memory, at first slowed in its action, fails. Some patients become talkative and querulous, though the majority of them remain placid and good-tempered; but many develop a tendency to nurse unwarrantable suspicions about their friends and attendants. Insanity, or a condition bordering upon it, not unfrequently develops; occasionally the mental alteration manifests itself in suicidal tendencies, or passes into one of complete dementia as hebétude, listlessness, torpor, and lethargy progress.

In the early stages hallucinations of sight and hearing are often present, and may remain.

The pulse is usually weak, and body temperature is always sub-normal; the patient complains of great discomfort when the weather is cold. The gait is slow, like all other movements, and some apparent failure of coördination is often present, which causes the patient to stagger, trip, and even to fall at times.

The condition of the viscera usually indicates nothing abnormal, but in the final stages albuminuria or glycosuria may be sometimes observed; the retarded metabolism shows itself in a diminution of the amount of urea excreted. There is a tendency to hæmorrhages after trivial injuries, which may become a serious complication after delivery, should the myxœdematous patient become pregnant. More or less anæmia is a constant feature.

The course and duration of the disease vary greatly; often there is a period of prolonged weakness or lassitude before the characteristic symptoms show themselves. The disease, once established, is progressive, though it may appear sometimes to halt or resolve itself for a time during pregnancy. If untreated, the patient may exist in his dull lethargic way for years, unless cut off by some intercurrent affection. Death should never now be witnessed from myxœdema, since the treatment is invariably successful as long as the proper remedy is used.

Pathological Anatomy and Pathology.—Nothing has been stated in the above description of the signs and symptoms of the disease about the condition of the thyroid gland, because usually it is difficult to be certain about this during life. The gland is nearly always wasted and diminished in weight; very rarely is it increased in size, and sometimes it has so withered as to be recognised with difficulty. The pathology of the affection is unquestionably to be fully explained by the theory of the absence of the function of the thyroid gland; it supervenes upon its destruction by disease and surgical removal in man and animals, and the symptoms disappear entirely upon the administration of the gland-substance or its active principles. In those cases of myxœdema where the thyroid is enlarged there is always evidence that the increase is caused by some fibroid or other change which has destroyed the secretory elements of the gland. Gley, Vassale, and Welsh maintain that the nervous or mental symptoms are the result of loss of function of the parathyroid or accessory thyroids, and that the trophic disturbances are caused by loss of thyroid activity.

The changes found in the skin correspond to those observed during life; the solid œdema is the result of proliferation of its fibrous and nuclear elements; the sweat-glands and hair-follicles are wasted partly owing to the pressure of the new fibres. Mucin has been found in abnormal quantity in the skin and other tissues, though it is usually absent in the last stages of the disease. The liver, kidney, heart, and vessels in advanced cases may exhibit cirrhotic changes of the same nature as those observed in the connective tissue of the skin.

Diagnosis.—The disease, when fully developed, can scarcely be mistaken, but the writer has witnessed several cases in

which impairment of mental power, without any of the other symptoms or signs of myxœdema, suggested a diminution in the activity of the thyroid functions, which entirely disappeared upon thyroid feeding. There can be little doubt that many cases escape recognition in this early stage.

Chronic Bright's disease is easily eliminated by the recognition of absence of pitting and by the state of the urine, though in advanced myxœdema there may be albuminuria, in which case, however, the other symptoms will be unmistakable.

Acromegaly is differentiated by the enlargement of the hands, feet, and face being due to true bony hypertrophy. In *Leontiasis* the bony hypertrophy is confined to the head and face, whilst in *Gigantism* the condition begins in childhood and affects all the bones, and, moreover, in these rare affections the characteristic mental and speech symptoms are absent.

Prognosis.—The disease is a progressive one, and if untreated will end in death from inanition, hæmorrhages, tuberculosis, or other intercurrent affection, or death may result from chronic heart-changes. The prognosis is, however, always very good, save in neglected cases of long standing, where myocardial or renal degeneration has developed, and even such cases usually markedly improve. In one sense the disease must be regarded as incurable, since thyroid feeding must be maintained during life; but in the great majority of cases a perfectly healthy standard may be maintained till old age by the administration of the remedy.

Treatment.—The principles upon which thyroid feeding should be carried out are detailed under 'Cretinism.' The dose in myxœdema at first should be larger than that which ultimately will be necessary to keep the individual in perfect health. Ten minims of the B.P. liquor may be taken as equivalent to about the tenth part of a sheep's thyroid, and are almost equal in activity to two grains of thyroideum siccum. This amount may be given daily at bedtime till the normal condition of health is reached, but as a rule it will be well to commence with half the above dose, which may be gradually increased during the first week. In six or eight weeks in mild cases, and in double this period in severe cases, many of the symptoms of the disease may have disappeared, but the baldness continues for a much longer time, though marked improvement in the growth and condition of the hair may be observed in three or four weeks. The facial aspect changes, the mental symptoms and deliberate speech-characteristics disappear, and in a few months it is not unusual for the patient to find that she is not recognised by her intimate friends, so great is the improvement. Relapse is certain unless the drug be continued at this stage. Murray, to whom is due most of our practical acquaintance with the mouth-

administration of the thyroid remedy, calculates that sixty minims per week of the liquor represent the output of a healthy thyroid gland in the human subject, and this amount may be continued for an indefinite period. The writer, in his cases, found that half this dose—viz. 5 mins. every night, or 6 grs. of the dry thyroid once a week—was usually sufficient to maintain the healthy standard. Upon page 253 the methods of regulation of the dose—viz. the weighing, pulse and temperature recording, are referred to, but intelligent patients soon learn to graduate the necessary amount of the drug to their requirements.

It is advisable to see that the dried gland and the liquid preparation are in a proper state of preservation; there is no advantage in resorting to the various preparations supposed to contain the isolated active principles of the thyroid. As the disease is now recognised at an early stage, the large doses are seldom necessary which were formerly employed in the reported cases, the majority of which were in a very advanced condition when first met with.

NEPHRITIS. (Sec under BRIGHT'S DISEASE, p. 110.)

NEPHROLITHIASIS AND NEPHROPTOSIS.
(See under KIDNEY.)

NEURALGIA

The term is applied to a severe pain, generally of a paroxysmal nature, experienced in the course of a nerve-trunk or its branches, and supposed to be of functional origin.

Etiology.—Children are seldom affected, the condition being one met with most frequently in middle or past middle life. Males and females suffer about equally. A neurotic history is obtainable in a large proportion of cases. All exhausting diseases or conditions act as exciting or predisposing causes; thus it is common as a result of anæmia, however induced, and is seen in its typical form during prolonged lactation, pregnancy, and after the severe hæmorrhages which often precede the climacteric. Intense and prolonged mental strain and emotional disturbances are a fruitful source, especially if the former cause be associated with errors in refraction. The condition is often seen to accompany or follow the eruptive fevers, influenza, malaria, rheumatism, Bright's disease and arterio-sclerosis, gout, and diabetes. In these cases the etiological factor is obviously a toxin; and poisons, as lead, copper, arsenic, mercury, or alcohol, may act in a similar

manner. Exposure to cold, especially if localised, and trauma, are frequently seen to act as exciting causes. Reflex irritation originating in the peripheral extremity of a nerve is a very common factor in facial neuralgia, where caries of teeth or disease of the antrum and frontal sinuses may set up an intense pain along the branches of the fifth nerve. Organic diseases which directly involve the nerve-trunks, as tumours, caries of bone, meningitis, &c., produce pains of a neuralgic character; these, however, do not strictly fall under the above definition of neuralgia. For the same reason neuritis, or inflammation of a nerve, is not included, though the discrimination between it and neuralgia is sometimes almost impossible.

Symptoms.—The most characteristic feature in the pain of neuralgia is its paroxysmal nature; the attacks last from a few minutes to as many hours. The pain during this period may not be continuous in the strict sense, being of a stabbing, darting, jumping nature, though sometimes during the paroxysm it is described by the patient as a dull continuous aching; but even in this latter class of case it is liable to violent exacerbations. As described by Gowers, a typical attack consists of a series of sudden sharp pains occurring every few minutes, the series constituting a 'paroxysm,' and a series of these constituting an 'attack.'

The pain is *spontaneous*, but is very liable also to be brought on by movement, coughing, sneezing, exposure to cold, or heat, emotion, pressure, or other causes. In the intervals between attacks all sensation of pain may be absent, but there is usually some abnormal sensation which causes the patient to state paradoxically that, though he feels no pain, he 'knows that it is there.' Numbness, tingling, or transient anæsthesia may follow an attack. Trophic, motor, vaso-motor, and secretory changes may be observed during or after a severe attack in the region affected. Thus there may be blanching of the hair, twitching of muscles, flushing of the skin, localised sweating, œdema, or erythema, and, in facial neuralgia, lacrymation or salivation. Even thickening of the bones and periosteum has been observed in long-standing cases. The pain is usually unilateral, involving the region supplied by a particular nerve, darting sometimes in a peripheral and at other times in a centrifugal direction, usually being experienced as deeper than the cutaneous level, and sometimes radiating to adjacent nerves, and even transferred to the corresponding nerve on the opposite side.

There is usually marked tenderness or hyperæsthesia of the skin in the region involved in the spontaneous pain. Certain points exhibit special tenderness; these in the main correspond to the spots at which the nerve-trunk or its branches emerge from bony canals or openings in the fascia, and are generally present in all cases of long standing. Pressure upon

these points usually brings on a paroxysm, and in spontaneous paroxysms the pain is generally more intense in their immediate vicinity, and then even slight pressure over them exaggerates the acute agonising pain. Tenderness over the vertebral spines corresponding to the affected nerves is not uncommon.

The intervals between the attacks are most variable, though sometimes they may be of a constant length, constituting a remarkable periodicity, which is the rule in the neuralgias occurring in the malarial cachexia; but this feature is by no means confined to the malarial condition.

Pathology.—Though minute changes have been sometimes observed in cases of long standing, idiopathic neuralgia must, in our present state of knowledge, be regarded as a purely functional affection. The generally accepted explanation of the origin of the pain is that it is not situated in the nerve-trunks, their branches, or their terminal nerve-organs, but that the disturbance of function is confined to the central sensory nerve-cells within the spinal cord, in which the sensory nerve-fibres end or originate. The nature of the central disturbance of function may be regarded as an over-action with spontaneous discharges of nerve-force from the affected cells, probably from those situated in the grey matter of the posterior cornua, and not in the ganglia of the posterior nerve-roots. Of the causes which produce the unstable equilibrium of these cells nothing can be said to be definitely known.

NEURALGIA OF THE FIFTH NERVE

This is also known as Trigeminal and Trifacial Neuralgia, Tic Douloureux, and Prosopalgia, and is by far the most common form of neuralgia.

Etiology.—The etiological factors are those already stated in the general description of neuralgia. It is, however, necessary to emphasise here the frequency with which the disease is due to *reflex* causes, as seen in connection with disease of the teeth and their alveoli, frontal sinuses, nasal fossæ, middle ear, eye, and orbit. Some of the worst cases may, however, be observed in patients whose jaws are completely toothless, and in whom no reflex source of irritation can be discovered.

Symptoms.—These vary according to the branch of the nerve chiefly involved, and in severe cases all the divisions of the fifth may be the seat of pain of the most terrible intensity, constituting the rare so-called *epileptic* or *quintus* neuralgia, which sometimes drives its victim to self-destruction in order to escape the unbearable agony caused by the frequent paroxysms of the disease. The affection, except in diabetes, is seldom bilateral; usually only one or two divisions of the nerve suffer.

Affection of the *first division*, known as *supra-orbital neuralgia*, is commonly confined to the supra-orbital branch; it is very frequently observed in the malarial cachexia, and as a sequel to influenza. The pain darts from above the eye in the region of the notch, and radiates over the temple, side of the cranium and anterior half of the vault to the coronary suture. The eyelid and side of the nose, and the eye itself, are also the seat of severe pain when the entire first division is involved (*ophthalmic neuralgia*), as it often is in frontal sinus trouble. The pressure-point in supra-orbital neuralgia is immediately above the notch, but other points may be determined—viz. one on the upper eyelid, another over the lower edge of the nasal bone, and sometimes on the globe itself.

The pain may be limited to the eyeball (*ciliary* or *ocular neuralgia*) and be associated with photophobia, lacrymation, conjunctival suffusion, and some amblyopia. This variety is often due to errors in refraction, conjunctival or corneal disease, and glaucoma, and the pain may also affect the opposite eye, especially after severe exercise of the eyesight.

Neuralgia of the *second division* (*supra-maxillary neuralgia*) usually follows the distribution of the infra-orbital branch, and the chief pressure-point is over the infra-orbital foramen; when the entire division is involved, points over the temple, side of the nose, upper lip, malar bone, and a tender line along the upper gum are usually also present. The pain is felt deep in the face and in the upper jaw and nose. The superior dental branch may be alone affected.

With involvement of the *third division* (*infra-maxillary neuralgia*) the pain is widely distributed over the temporal region, ear, lower jaw, chin, and tongue, when all its branches are affected. More commonly the alveolar twigs, the auriculo-temporal, or the lingual branch are alone involved. The chief pressure-point is over the inferior dental foramen; other points may be found over the temporal and parietal bones, in front of the ear, or in the tongue itself.

The spontaneous pain in all varieties of facial neuralgia may be intense or mild. The paroxysms are excited by cold, pressure, emotional excitement, movements of the jaws and facial muscles, and in severe paroxysms the pain commencing in one region may radiate through all the divisions of the nerve, and also extend over the occiput, down the neck, to the shoulder. In the true *tic douloureux*, or epileptic neuralgia, the remissions, at first variable in their duration, tend to shorten as the disease progresses, and the paroxysms, which may last but a few minutes, tend to become more intense in their severity, till the patient can scarcely be said to be quite free from them, or free for but a few minutes at a time, all the divisions being usually finally involved. The vaso-motor, trophic, secretory,

and spasmodic phenomena already described may be present in any case; facial hemiatrophy sometimes occurs, herpes is not unusual, flashes of light and transient paralysis of the third nerve may be experienced.

Facial neuralgia may last indefinitely; in the writer's experience the disease is most intractable in diabetic and glycosuric conditions occurring in senile patients. The paroxysms which are brought on by attempts at chewing and swallowing cause serious interference with the general nutrition of the body; muscular weakness supervenes, which, when associated with insomnia, may seriously reduce the condition of the patient.

CERVICO-OCCIPITAL NEURALGIA

The pain is felt in the region supplied by the upper four cervical nerves, but most frequently in the area of distribution of the great occipital. The back of the neck and posterior aspect of the head up to the vertex is the seat of an almost continuous dull pain, which is frequently bilateral, with occasional paroxysms or exacerbations of a more acute or severe type, caused by movements of the head, sneezing, or coughing. The pressure-point in occipital neuralgia is situated over the spot at which the great occipital emerges—viz. about midway between the mastoid process and the spine; usually there is a tender spot over the parietal eminence. The neck is stiff, so that cervical caries is simulated; sometimes, though rarely, the lymphatic glands are enlarged; the skin over the posterior region of the scalp and neck is usually hyperæsthetic, and the pupil may be contracted; local baldness sometimes follows. Exposure to cold, trauma, and influenza are the usual causes of this affection.

BRACHIAL or CERVICO-BRACHIAL NEURALGIA

In this variety the region innervated by the lower four cervical with the first dorsal is the seat of pain. The lower part of the neck, shoulder, or axilla suffer; more frequently the area of distribution of the ulnar and musculo-spiral, may be also involved, causing pain in the arm and hand. The character of the pain is burning or boring, and it may approach to the continuous type, but more frequently it is experienced in darting paroxysms which are always induced or intensified by some movement of the arm or hand. The patient instinctively supports the affected limb, so as to prevent all motion or vibrations.

The pressure-points are numerous and variable, the most important being over the musculo-spiral where it winds round the humerus, over the ulnar between the olecranon and condyle, over the same nerve on the anterior aspect of the wrist, and over the circumflex at the posterior margin of

the deltoid where it gives off its cutaneous branch. Pressure on these points brings on a paroxysm of pain preceded by numbness and formication, symptoms which may be also present in the intervals between the attacks. Tenderness may often be elicited in spots close to the lower cervical spinous processes.

This variety of neuralgia is very frequently the result of trauma; it is sometimes of influenzal or rheumatic origin; it is not rarely seen in anæmic subjects who are hysterical. The pain may radiate to the chest-wall and be mistaken for angina, but on the other hand organic heart-affections, sub-clavian and aortic aneurisms, may produce a condition almost identical with brachial neuralgia. Caries of the vertebræ, meningitis, and tumours are also liable to produce similar symptoms. Oppenheim regards true brachial neuralgia as a rare affection, most of the cases being of the nature of a *brachialgia* or pain in the arm of indefinite localisation, which is but the manifestation of a general neuropathic condition associated with depression, insomnia, or other psychopathic symptoms.

INTERCOSTAL NEURALGIA

The pain, which approaches to the continuous type but is subject to acute exacerbations, is experienced in one or several of the intercostal spaces innervated by the anterior branches arising from the dorsal roots. Occasionally it is bilateral, and it may extend upwards to the shoulder or downwards along the inner aspect of the arm. The pressure-points are especially important in the diagnosis of this variety of neuralgia. They correspond to the spots at which the three branches of the intercostal nerves become superficial—viz. close to the spine behind, at the side of the sternum in front, and laterally midway between these in the line of the axilla.

Mastodynia, or neuralgia of the nerves of the mammary gland, is common in women. It is associated with severe paroxysmal pain in the breast, with hyperæsthesia of the skin and tenderness of the nipple.

Gowers regards *pleurodynia* as a true neuralgia of the *pleural* nerves, which differs from intercostal neuralgia by being limited to a particular space independent of the distribution of any intercostal nerve or nerves.

Intercostal neuralgia is often associated with herpes zoster, though most authorities regard the pain in such cases as being the result of a neuritis. Intractable intercostal neuralgia may follow in one or more intercostal spaces long after the disappearance of herpes zoster.

The disease is most common in anæmic, hysterical, or neurotic women. It may follow trauma and exposure to cold, and is

not rarely associated with manifestations of rheumatism or neuralgia elsewhere.

It is more frequent upon the left side and is sometimes associated with disturbances of the cardiac rhythm ; a severe paroxysm is liable to be mistaken for angina, with which it may also be associated. It may simulate (when situated low down) the rare form of neuralgia which affects the *phrenic nerve* especially in pleural and pericardial mischief, but the distribution of the pain of phrenic neuralgia corresponds in the main to the line of the level of the attachments of the diaphragm.

LUMBAR AND ABDOMINAL NEURALGIA

Correspond to the intercostal form. The most common variety, known as *lumbo-abdominal*, affects the distribution of the ileo-inguinal, hypogastric, and the branches of the genito-crural nerves, causing radiating and often bilateral pains in the lower zone of the abdomen, groins, perinæum, scrotum, and testicle. The pressure-points are located above the middle of the iliac crest, beside the lumbar spines, at the lower part of the rectus, and in the scrotum or labium. The paroxysms are excited by walking, and the distribution of the pain is liable to much variation in different attacks in the same individual. In women it is not rarely caused by pelvic mischief. Neuralgia of the *coccygeal* nerves is separately described under 'Coccydynia' (p. 223).

CRURAL NEURALGIA

In this form the long lumbar nerves are involved—viz. the crural, obturator, and anterior branch of the external cutaneous of the thigh. The pain radiates along the lines of distribution of the nerve affected, the most frequently observed variety being experienced along the outer surface of the thigh as low as the knee. *Obturator* neuralgia is felt in the course of the nerve from the foramen to the knee-joint in the adductor muscles along the front of the thigh. In the writer's experience it is more frequent than most authorities recognise ; he has seen it several times mistaken for hip-joint disease and rheumatoid arthritis. The pain is sometimes only experienced during walking and for a short time afterwards. It may be caused by involvement of the nerve in an obturator hernia. All varieties of crural neuralgia are liable to be closely simulated by the pressure of tumours and organic lesions which cause neuritis. The pressure-points are of considerable importance in the diagnosis ; they are situated over the branchings of the different nerves involved.

Sciatica is by far the most common form of painful affection of the nerves of the lower extremity. It is usually due to

acute neuritis of the great sciatic, but a true neuralgic form is not uncommon. It will be found described, for convenience of reference, under its own heading.

Neuralgia of the feet is very rare, most of the cases so described being examples of neuritis. Flat-foot causes great pain by the stretching of the fibrous structures supporting the arch, and it should not be confounded with so-called 'plantar neuralgia,' which is always a form of neuritis.

Achillodynia, or pain at the insertion of the tendo Achillis, is never of true neuralgic origin, but is due to inflammation of the neighbouring bursa.

Painful Heel is probably due to neuritis. There is great tenderness over the plantar surface on standing or on digital pressure. In a typical chronic case seen by the writer, neuritis of a cutaneous nerve of the arm followed the recovery from the heel-pain.

Metatarsalgia, as described by Morton, is supposed by some to be due to neuralgia of the external plantar nerve. It is more likely to be caused by neuritis induced by pressure of the branch supplying the fourth metatarso-phalangeal joint.

Intermittent claudication, or intermittent limping, as described by Charcot, is sometimes mistaken for neuralgia of the leg-muscles. It may be closely simulated by obturator or crural neuralgia. There are tingling, numbness, marked pain and muscular weakness in one leg on commencing to walk, which sometimes renders progression impossible without frequent resting and halting. This diseased condition is regarded as the result of degeneration of the coats of the large arteries of the leg, which are thereby unable to supply blood to the working muscles; there may also be an element of vascular spasm in its pathology.

SPINAL-COLUMN NEURALGIA

Many authorities regard the painful condition of the spine following railway accidents as of a true neuralgic nature. There is generally severe pain, increased by movements of the body, especially vibratile motions as in carriage-driving. Marked tenderness over a considerable vertical extent of the spine is usually present, especially in the dorsal region. The pain is seldom distinctly paroxysmal. The condition seems apparently identical with that which occurs in the spine in hysterical or other patients suffering simultaneously from some form of true neuralgia in other parts of the body. The suddenness with which it may entirely disappear upon the termination of legal proceedings suggests to the writer that it may be but a local manifestation of a general psychopathic condition in many cases.

VISCERAL NEURALGIA

That a true neuralgic affection of the viscera may occur cannot be doubted, but the diagnosis of a purely functional affection of the visceral nerves can only be regarded as likely when all forms of organic disease can be eliminated. Under 'Angina Pectoris,' 'Gastralgia,' and other neuroses, several forms of visceral neuralgia are described.

Hepatalgia, Enteralgia, Nephralgia, Ovaralgia, &c., are titles employed when a painful condition of the liver, intestine, kidney, or ovary exists without evidence of organic disease or disturbance of function of these viscera. The disease is usually met with in hysterical or neurasthenic women. The character of the pain in the main corresponds with that observed in other forms of neuralgia—viz. it is usually of paroxysmal nature and may be entirely absent between the attacks; though usually spontaneous, paroxysms may be induced by such pressure as it may be necessary to employ in palpating the affected organ. Tenderness is an almost constant feature, and is easily elicited by moderate pressure over the ovary when this organ has its nerve-supply involved, as is almost constantly seen in hysteria. The attacks of pain in neuralgia of the kidney may closely resemble those produced by calculi. In visceral neuralgia the pain is often found to be reflected along certain peripheral nerves corresponding to the spinal-cord segment which receives the fibres of the sympathetic supplying the affected viscus.

Diagnosis.—The main points in the differentiation of the various forms of neuralgia have been briefly referred to in the description of each, and need not be repeated. The general characters of neuralgic pain must be always remembered in the exclusion of organic diseases in all cases of doubtful diagnosis. Thus it corresponds in distribution to a particular nerve or nerves, and it is always unilateral save in the exceptions above detailed. The pain is intermitting or remitting, coming on in paroxysmal attacks. In one attack the spot of maximum intensity of pain may shift to another focus in the next paroxysm.

Neuritis produces a type of pain which is continuous, though there may be occasional exacerbations; there are no marked tender or *pressure points*, but the nerve is tender to pressure *throughout its course* when this is accessible to the touch of the finger, and it may be obviously swollen. Where the neuritis has been established for some time the muscles supplied by the affected nerve become flabby and waste, and more or less paralysis supervenes; their electrical excitability changes and the skin may become anæsthetic.

Organic disease affecting nerve trunks or branches seldom causes migratory pain; the symptoms are more constant,

complete intermissions being rarely observed, and mental or emotional excitement does not cause exacerbations of suffering.

Hysteria is eliminated by its own marked psychic features. The pain which hysterical subjects usually complain of seldom obeys the strict law of distribution characteristic of true neuralgia. It is always influenced by suggestion, emotion, or other psychic force, and occurs frequently in patches or segments, with its accompanying anæsthesia. It must not be forgotten that hysterical patients may suffer from true neuralgia.

Hypochondriasis is frequently associated with morbid sensitiveness in the visceral organs which may closely simulate visceral neuralgia, but the mental condition always betrays the characteristic introspection which is the cause of the abnormal sensations. The pains of *locomotor ataxia* are recognised by their lightning-like suddenness, their irregular distribution, and by the other signs of that disease.

Prognosis.—The danger to life may be regarded as nil, but the outlook is seldom bright save in mild acute cases. In the severe or epileptiform variety it is very gloomy: practically it means a life of misery unless formidable surgical measures be resorted to. As a rule, the prognosis in all cases becomes less favourable in proportion to the duration of the disease and to the absence of any easily corrected cause, as anæmia, over-work, &c. The prospect of a speedy and permanent cure brightens as a rule in proportion to the youth of the patient; senile cases are usually most obstinate. Glycosuric or diabetic cases are often intractable, and so also is the form of neuralgia that follows long after the disappearance of herpes zoster.

Treatment.—The removal of the neuralgic condition is a complicated problem, and much of the treatment for its relief must be still confessed to be largely empirical. In the author's 'Dictionary of Treatment,' upon pages 592–609, the relative values of the different drugs and remedial agents are discussed at considerable length; only a brief outline of the general principles which should govern the treatment need be mentioned here.

The first and most obvious indication is the removal of anything which can be regarded as the cause of the pain. In severe facial neuralgia, which is sometimes a *reflex irritation* from a diseased tooth, though the latter may exhibit little tenderness or pain in itself, its removal may give permanent relief. It is not unusual, however, to see neuralgic subjects who have had both jaws cleared of all their teeth without any benefit whatever. Anæmia calls for large doses of iron in combination with moderate amounts of arsenic. Digestive troubles, uterine or ovarian disease, syphilis, insomnia, glycosuria, gout, rheumatism, and other underlying conditions

which may be etiological factors in the case, must be treated upon the principles indicated in each. Everything which raises the standard of health should be tried, as improved dieting, fresh air, change of scene and often of occupation, with regulation of the hours of work, rest, and sleep.

For the relief of pain nothing can be compared with morphia when administered hypodermically, but it should never be resorted to in chronic and very seldom even in acute cases, owing to the danger of the establishment of the opium-habit. This is almost certain to follow if the patient be once permitted to use the syringe himself. The same rigid caution should be exercised in the use of cocaine, alcohol, Indian hemp, or other narcotic. The writer's method of deep parenchymatous injection of a very diluted solution of morphia lodged at several places in the trunk of the affected nerve or around its sheath, is a combination of acupuncture, aquapuncture, hypodermic injection, and anodyne medication, and may be safely tried for a few times at the beginning of the treatment; it is not only palliative, but sometimes a speedy and permanent curative effect follows, especially in neuralgic sciatica.

Drugs of the analgesic class which have little or no action on the cerebral functions are often invaluable, as antipyrin, phenacetin, exalgin, antifebrin, butyl-chloral hydrate, salol, salicylates, and bromides. Perhaps the most reliable of all this class of agents is aspirin in doses of about 15 grs. twice or thrice daily.

Quinine, which is almost specific in malarial cases, is often most beneficial in the non-malarial types of the disease; those who deny its value probably use it in doses which are too small: less than 15 grs. daily need not be tried. Chloride of ammonium, phosphorus, strychnine, zinc phosphide, and a host of drugs have been vaunted as specifics; undoubtedly most of them will occasionally give a satisfactory result, but there are as yet no clear indications for the selection of many of them. Gelsemium, aconite, and butyl-chloral are believed to possess a special action upon the trigeminal nerve, and bromides appear to have some special influence over the forms of ovarian neuralgia.

The value of iodides and salicylates is unquestionable in rheumatic cases and in the neuralgias occurring in the late stages of syphilis. Nitroglycerin may be empirically tried in any type of the disease.

Local agents for the relief of pain are almost innumerable; menthol, cocaine, antipyrin, aconite, belladonna, chloroform, and every known local analgesic have been recommended and occasionally found useful. Freezing by ethyl chloride or ether often gives temporary ease.

Counter-irritation by blistering, by thermo-cautery, or other method may be used, and electricity in different forms is

valuable. The injection of osmic acid into the affected nerve should always be tried before resorting to operative measures. These latter consist in *stretching* the nerve; *neurotomy* or simple section of its trunk; *avulsion* or tearing out of a nerve-trunk from its bony canal; *neurectomy* or the removal of a large portion of it; sometimes *total resection* is performed.

In severe trigeminal neuralgia Krause's operation of intracranial resection of the nerve and even of removal of the Gasserian ganglion with its posterior root, has been most successful in cases formerly beyond the reach of palliation.

NEURASTHENIA

This term is a synonym for nervous exhaustion or nervous debility—words which sufficiently define the condition as far as our present knowledge permits.

Etiology.—The disease occurs in both sexes, but in its typical form it is seen most frequently in males about middle-life. It is met with, however, at all ages, and its features may occasionally be observed so early as to justify the recognition of a congenital form.

Heredity plays the chief rôle in the etiology of the disease, and the liability of parents to hand down this condition to their offspring may be easily acquired by their living a too strenuous life which makes demands upon their nerve-force, often culminating in 'nervous breakdown.' Mott believes that syphilis, alcoholism, and tuberculosis in the parents at the time of conception may be the cause of inherited weakness in the nervous system of their offspring. The children of such parents inherit nerve-cells of an inferior capacity for the creation of the nerve-force necessary for the ordinary struggle for existence. Such subjects in their turn may hand down their inherited neuropathic weakness without having unduly drawn upon their limited supply of nerve-force.

The writer, judging from his own experiences, is led to believe that the worst forms of the inherited variety of the disease are met with in those subjects who have been born when their parents had already passed middle-age—*i.e.* when the causative factor of mental or psychic over-strain had been longest in action.

The *acquired* form of neurasthenia may be produced by but slight provocation in those subjects who have inherited the neuropathic disposition, but the most healthy and robust type of nervous system will inevitably succumb to any strain which is prolonged and intense. The great majority of all cases of acquired nervous exhaustion are, however, met with in those who have inherited an abnormally weak condition of the nervous system; consequently the strain which is

supposed too often to be the main etiological factor in their break-down is but the exciting cause.

Of these exciting causes a profound mental emotion, as the terror experienced when a serious railway or other accident seems imminent, or the shock caused by a real injury, or sun-stroke, may effect in a moment what is more frequently accomplished by a long period of mental over-strain. Such causes are frequently regarded as traumatic, but they differ little in their operation from those more commonly met with. These are prolonged mental over-work, especially when associated with worry, or when the element of struggling against time and competition of other sorts enters into the brain-labour, which must then be accomplished under dangerous high pressure. Hence the frequency with which nerve-exhaustion is seen in authors, journalists, public speakers, professional men, stock-exchange manipulators, and students preparing for examination. Sexual excesses, masturbation, and especially interrupted or perverted sexual intercourse, is an important etiological factor, so that a form of neurasthenia described as the *sexual* is often recognised. Toxic causes may be also at work; thus the condition is common after influenza and certain fevers, syphilis, chronic poisoning by lead, arsenic, tobacco, morphia, or alcohol. Probably toxæmia may be the explanation of the neurasthenia that sometimes follows disorders of the stomach or intestines in which auto-intoxication is supposed to occur. Some few neurasthenics may only exhibit their inherited weakness after undue demands have been made upon their muscular system, though the majority of patients suffering from nerve-exhaustion are capable of prolonged muscular exercises without experiencing anything but benefit thereby. The nerve-exhaustion sometimes seen in Glénard's disease is probably an accidental association.

Pathology.—Upon this one can only speculate, since the condition cannot be said to possess a morbid anatomy according to the methods of investigation at our disposal. It must be regarded as akin to, or identical with, the state of fatigue which follows all prolonged functional activity, the process of repair being faulty in neurasthenia.

Symptoms.—These are variable, and with the exceptions to be mentioned later on are entirely subjective. According to the portions of the nervous system which suffer most obviously, several varieties of the disease have been described, as the psychic or cerebral, the spinal, cerebro-spinal, sexual, visceral, &c.; but even in the most typical of these, evidence of some involvement of the entire nervous system is generally obtainable, though over-shadowed by the local symptoms.

The patient is generally, but not always, somewhat depressed in spirits, and he is usually irritable and easily excited, the

excitement being always characterised by a rapidly supervening sense of exhaustion, which lasts for a considerable period after the withdrawal of the stimuli which have caused the excitement. The *cerebral* form is the most common; the chief obvious symptom is the difficulty with which ordinary mental work is accomplished, especially in those who formerly were capable of prolonged and severe intellectual exercises. The mental powers preserve their due balance, and memory is usually intact; but the effort to recall facts, names, and incidents is associated with such a degree of exhaustion that the patient often refrains from exercising it, and complains that his memory is failing. Sometimes even the writing of an ordinary letter is difficult, and may be followed by prolonged fatigue or exhaustion, which shows itself in headache, flushing, or throbbing of the temples. More frequent than headache is the sensation of weight or pressure upon the brain, which is often described as if some heavy object were crushing the cranium. The scalp may be tender all over or in patches, and hyperæsthesia of the skin of the face about the orbits is often present. It is not unusual in the writer's experience for such patients to complain of a more or less constant sensation as if a postage-stamp were adherent to some spot upon the forehead, which spot upon examination is found to be the seat of neither anæsthesia nor hyperæsthesia.

Vertigo is often complained of. Insomnia is a usual symptom, and generally aggravates the condition by inducing a dread of complete sleeplessness. As the patient lies awake in a state of involuntary mental activity, having nothing else to occupy his thoughts, they are turned inwards upon his morbid sensations and he may become hypochondriacal, a condition usually escaped from by those neurasthenics who sleep well. Suicidal tendencies may be developed in this way, and a not rare symptom is the fear of becoming insane, which may also be accompanied by any of the following psychopathic conditions:

Anxieties, fears, or phobias of various kinds are present in most severe cases of neurasthenia—*i.e.* *nosophobia* or *pathophobia* (the dread of sickness), *phobophobia* (the fear of fear), *panophobia* (the fear of everything), *agoraphobia* (the fear of locality, often manifest in the dread of crossing open spaces), *claustrophobia* (the fear of remaining in a confined space), *anthrophobia* (the dread of being amongst people), *monophobia* (the fear of solitude), *astrophobia* (the fear of storms), *siderodromophobia* (the fear of railway travelling), *mysophobia* (the fear of contamination), &c.

Asthenopia is a very common symptom, the weakness or fatigue which follows the use of the eyes being due to exhaustion of the muscles of accommodation and of the recti. It is usually associated with sensations of flashes of light,

severe pain in the eyeballs, a dragging sensation behind the orbits or in the occipital region, and congestion of the conjunctiva—symptoms which are not relieved by the use of glasses and which only disappear on complete rest of the eyesight. The pupils are commonly normal or somewhat dilated and sometimes irregular in size, and an apparent ptosis from sympathetic involvement may be observed, but ocular paralysis does not occur. Hearing may be painful, and sometimes aural hallucinations and pulsating tinnitus are complained of.

In *spinal* neurasthenia the mental fatigue and exhaustion common in the cerebral form is replaced by fatigue felt in the muscles. This early exhaustion after even trivial exercise may be a marked feature in all forms of neurasthenia, but sometimes the muscular power and physical endurance are intact when mental exertion is followed by great depression and exhaustion. It is, however, rare for the spinal symptoms to be well marked without obvious symptoms of cerebral lag. Pain in the spine is more or less constant, and aggravated by exercise. Tenderness or hyperæsthesia in various parts of the column is common, with aching of the legs associated with a sense of stiffness, tingling, or numbness. These abnormal sensations are also experienced in the viscera or other parts of the body, and may develop into or simulate true neuralgia.

The tendon-reflexes are increased, but ankle-clonus does not occur; the muscles respond more readily to mechanical stimuli than they do in health, and rapid vibratory or fibrillary tremors are not uncommon even in the facial muscles. These constitute the main *objective* symptoms or signs met with in neurasthenia, and hence are of considerable importance in diagnosis; with them signs of cardiac and vaso-motor disturbance may also be observed. When the condition is extreme, paralysis may be suspected, but this never occurs, nor is the weakness ever confined to any particular muscle-group, though in very rare cases it may simulate incomplete hemiplegia.

In the so-called *visceral* type of the disease, in addition to many of the above symptoms, various pains or discomforts, amounting sometimes to disorders of function, are observed. The heart may be irregular, quick, or slow in its action; more frequently palpitation is the most obvious symptom in the condition sometimes described as *neurasthenia cordis*; and even anginiform attacks may be noticed.

In stomach-cases, dyspeptic disorders may overshadow the general condition, the symptoms being identical with those seen in the various forms of gastric neurosis. the most serious being a complete anorexia which may in rare cases be the only obvious manifestation of the general neurasthenic state.

Constipation is the rule. These cases ultimately eventuate in profound emaciation and debility.

The urinary organs are often the seat of various disorders. Nervous ischuria may be marked and the amount of urine passed may be greatly diminished, falling at times to less than half a pint in the twenty-four hours; more frequently there is pollakuria—a nervous desire to pass urine very often in small quantities, especially when the patient feels that his surroundings do not afford a convenient opportunity for the emptying of the bladder. Oxaluria, lithæmia, or polyuria may also be present; albuminuria is rare, but small quantities of sugar may appear for short periods.

In *sexual* neurasthenia there is often the history of early masturbation which preys upon the patient's mind so that the dread of impotence becomes a serious symptom. The psychic element may be so strong that real impotence not uncommonly occurs, but it is usually only transient. It may lead to suicidal impulses, and the mental condition is further aggravated by frequent emissions and a genuine spermatorrhœa after urination or defæcation, though the discharge in the majority of cases is only of prostatic origin. Such patients may present the clinical picture of an aggravated form of hypochondriasis grafted upon the underlying neurasthenia which has been directly produced by the nervous worry, the result of their prolonged mental anxiety about the sexual disturbances, and, amongst such, sexual perversions of various kinds may be met with. In women sexual excesses are seldom the cause of neurasthenia, but ovarian tenderness and pain and pelvic discomforts are not infrequent manifestations of the condition; pruritus ani or vulvæ are common symptoms.

The vaso-motor symptoms met with in neurasthenia are of importance, since they afford objective evidence of the abnormal condition. The most common of these is a morbid flushing of the skin of the face, neck, and chest, not unlike that occurring in women at the climacteric. It is always aggravated by consciousness or psychic disturbances, and may alternate with pallor or a bluish discoloration of the extremities. Slight scratching of the skin may produce marked urticaria or wheals (these may, however, occur spontaneously in neurasthenia). The phenomenon known as dermatography may be observed, in which, owing to the vaso-motor excitability, a word traced on the skin by a blank pencil shows out in vivid redness for a time. Alopecia may be observed, and the teeth decay often with rapidity. Visible exaggeration of the pulsations of the aorta and large vessels, and even capillary pulsations, often accompany the cardiac disturbances, which latter may be also included amongst the vaso-motor phenomena. Neurasthenic patients as a rule are markedly

influenced by changes of temperature : localised sweating may often be observed. The course and duration of the affection will be referred to under Prognosis.

Diagnosis.—The first step is the exclusion of *organic diseases*, which can only be effected by a careful examination and search for the physical signs and symptoms of such organic nerve-diseases as *disseminated sclerosis*, *tabes*, *spinal meningitis*, *brain-tumour* or *brain-syphilis*, and *general paralysis*. The visceral form may so closely simulate *heart-disease* that a diagnosis between *neurasthenia cordis* and the organic and the functional forms of cardiac distress may be difficult. The organic heart-lesions are more easily excluded, because even should murmurs be present, as they sometimes are in *neurasthenia*, there is never evidence of the compensatory changes and hypertrophy seen in valvular affections. The murmurs heard in the *neurasthenic* heart are either systolic or cardio-respiratory, and are often only audible during a paroxysm of excitement or palpitation. Of the functional heart-conditions the one most likely to be confounded with *neurasthenia* is that associated with *Graves' disease* before the *exophthalmos* has become evident, the main symptom being *tachycardia*, but even in this condition the constancy of the rapid action of the heart is against *neurasthenia*, whose symptoms are ever varying from day to day. This latter feature is also a strong point in the exclusion of all organic disease, whether of the nervous system or viscera. The throbbing of the arteries, water-hammer pulse, and capillary pulsation of *neurasthenia* are easily distinguished from aortic regurgitation by the absence of the murmur and hypertrophy.

The multiplicity of the symptoms, as narrated by the *neurasthenic* patient, is in the majority of cases quite sufficient to exclude any single organic disease, but the physician must be always upon his guard not to overlook any organic lesion which may be possibly present in the victim of nerve-exhaustion.

The greatest difficulty in the diagnosis lies in the exclusion of *hysteria*, and too often these neuroses are confounded because they may exist together, especially in women. The typical hysterical convulsive paroxysm, or the presence of the characteristic stigmata, especially the paralyses, anæsthesia, contractures, limitation of the visual field, aphonia, &c., are not met with in pure *neurasthenia*. The changes in the patient's character, especially the yearning for sympathy, and the instability or irritability exhibiting itself in the violent emotional disturbances, are in marked contrast to the mental and muscular conditions in the *neurasthenic*, which manifest themselves only in the weakness, fatigue, or complete exhaustion after exercise of function. The character of leaden pressure or weight, which is associated with the headache

constantly present, or following all mental exercises in neurasthenia, is so very different from the circumscribed clonus of hysteria as to be almost pathognomonic.

Hypochondriasis may be present in the neurasthenic patient, but in its pure form its existence is demonstrated easily by the detection of some actual delusions in connection with his symptoms.

Prognosis.—The course of the disease may be rapid, especially in the traumatic cases, as in those occurring in railway accidents, sunstroke, &c., when the symptoms may develop within a few days. In those which result from chronic mental strain, many months may elapse before the affection shows itself in its fully developed condition. In the acute cases, especially in the young, recovery may be rapid and complete, but here, as in every case of neurasthenia, the prognosis depends largely upon the degree of hereditary weakness. Thus, in hereditary neurasthenia, without any obvious exciting cause, the prognosis is bad as regards the fitness of the patient to maintain his struggle for existence. Much will obviously depend upon his environment and position, especially as regards his command of means enabling him to live a life free from mental drudgery of all kinds. The longer the symptoms of neurasthenia have been in existence, and the longer the period of strain before showing themselves, the less favourable is the prognosis. The outlook is good where the nature of the strain has been emotional; one often sees rapid and permanent recovery in the profound nervous exhaustion which follows the duties of nursing and tending a relative in an acutely fatal illness, but if the illness has been a chronic one the survivor's nervous neurasthenia may be permanent. Oppenheim maintains that cardiac neurasthenia may cause death by inducing atheromatous changes through the continual emotional excitement resulting from the fear of heart-disease. The development of hypochondriasis, or other mental state, obviously deepens the prognosis, and the appearance of suicidal tendencies is always a grave omen. Notwithstanding all these considerations, in the main the prospects of recovery are good, provided the patient be able to afford himself a prolonged rest and such change in his pursuits in the future as shall not entail the liability to further mental strain.

Treatment.—In the main this will correspond with that indicated in hysteria, though the conditions, as already stated, must not be confounded. Prophylaxis is of vital importance, and the obvious indications for the upbringing of the children of neurasthenic parents need not be dwelt upon. The choice of their life-work should not be decided upon without serious consideration; those callings or pursuits which entail continuous mental application or intellectual work liable to be demanded

under conditions of high pressure, even in short spells, must be prohibited.

When the symptoms have already appeared the urgent indication is rest, and the great majority of cases require nothing else. The rest, to be of use, must, however, be prolonged, and must be suitable to the peculiarities of the patient. The neurasthenic patient suffering from mental over-work is usually incapable of taking *complete* rest. With the exhaustion after mental effort there is usually associated a restless desire for more work, and enforced idleness often intensifies this. The secret of success in the treatment is the judicious alternations or changes in the nature of the patient's intellectual pursuits, which will minimise the tendency towards introspection and study of his symptoms. He must be tempted to interest himself in matters which will prevent the concentration of his mind upon his former labours as well as upon his feelings of incapacity, whilst at the same time every factor in the cause of his breakdown should be removed when possible. Hence the value of a long sea-voyage or prolonged trip amongst country scenes unfamiliar to the patient, busy cities being avoided. Sports and muscular exercises are invaluable to those whose physical condition warrants them, but enforced muscular activity may be quite as injurious as mental over-work to some patients, especially in the case of women. Golf is a haven devoid of danger to most over-worked professional men, and fishing or shooting is a similar boon to patients of weak muscular powers. Manual labour, active sports like football or cricket, farm work, and even mountain-climbing, may be indicated in neurasthenia caused by masturbation.

In all cases a careful regulation of the hours of rest and activity is an essential point. Insomnia, when it fails to respond to such treatment, must be met by a resort to drugs, and of these the simple hypnotics of the bromide, trional, sulphonal, or paraldehyde class must be selected in preference to narcotics, which for the most part should be prohibited. Symptoms such as dyspepsia, headache, palpitation, constipation, &c., are to be treated on the recognised lines suitable in ordinary conditions; hydropathy, massage, electrotherapy, and a carefully selected dietary from which tea, coffee, and alcohol are excluded, such dietary as can be obtained in suitable sanatoria, are all that these symptoms demand. Drugs like quinine, iron, arsenic, phosphorus, and strychnine are seldom indicated, though frequently prescribed.

The period of modified rest briefly sketched above, to be of any lasting or curative value, must be extended in most cases of the fully developed condition to at least six months. It must not be omitted that it is essential to take the neurasthenic patient fully into the confidence of the physician, who should

explain to him the nature of his ailment. The calm assurance of the absence of all organic disease, as in some of the minor forms of hypochondriasis and hysteria, sometimes produces an equally remarkable beneficial effect in neurasthenia.

Where the disease has been fully established for any considerable time, and where nutrition is seriously affected, as seen in the extreme emaciation of the patient, it will generally be found advisable to resort at once to a two months' course of the Weir-Mitchell treatment—viz. absolute rest, isolation, massage, forced feeding, and electropathy, after which a prolonged holiday trip must be prescribed.

NEURITIS

The term implies inflammation of a nerve, and the condition is met with in two well-recognised forms—*mononeuritis*, in which a single nerve is affected, and *multiple* or *polyneuritis*, in which many nerves are involved. This latter form is also frequently described as *multiple peripheral neuritis*.

Etiology.—In *mononeuritis* the cause is usually trauma: blows, wounds, pressure, dislocations of joints, fractures of bones, or inflammation of neighbouring bony or other structures often excite inflammation in the nerves in the immediate vicinity. Trauma, even when slight, may excite neuritis of a single nerve in alcoholic subjects or in those suffering from other organic or from metallic poisons. The pressure of muscles when violently contracting, and the compression by tools or implements employed in the various handicrafts, may cause inflammation of any nerve. Crutch-paralysis is a common example of traumatic neuritis, and the pressure of syphilitic, malignant, aneurismal, or other growths acts in a similar manner. Hypodermic injections of ether and osmic acid are liable to excite inflammation of a nerve trunk or branch in the vicinity of the puncture.

Exposure to cold is a common cause of mononeuritis, as often seen in the so-called rheumatic inflammation which affects the facial nerve when an individual has been exposed to a current of cold air playing upon the side of the head. As causative factors must also be included all those agents which usually induce multiple peripheral neuritis; thus the poison of influenza, puerperal sepsis, gout, &c., may only involve a single nerve, though this must be considered as a comparatively rare event.

The etiology of *multiple* or *peripheral neuritis* is always toxic: alcohol, lead, zinc, copper, arsenic, tobacco, gout, diabetes, influenza, rheumatism, gonorrhœa, leukæmia, erysipelas, diphtheria, beri-beri, leprosy, the poisons produced by the microbes of the exanthemata, typhoid and scarlet

fevers, and small-pox may all produce a general or polyneuritis. The poisons of puerperal fever, malaria, syphilis, parotitis, tuberculosis, and cerebro-spinal meningitis, as well as toxic agents like naphtha, carbon disulphide and dioxide, ether, aniline, and ptomaines sometimes, though rarely, cause polyneuritis. Various senile conditions, anæmias, and cachexiæ are often associated with a general inflammation of the peripheral nerves; and these, with the form which sometimes follows exposure of the entire body to extreme cold, are probably also of toxic nature. A combination of toxic agents is not uncommon, the most frequent being alcohol and arsenic, as recorded in the epidemic of neuritis caused by drinking arsenical beer; alcohol and tubercle, gout and lead, also are often combined etiological factors.

The disease, in either the mono- or polyneuritic forms, may appear at any age, but the latter mentioned type is rarely met with till middle-life, the exception being in the diphtheritic variety, which is often seen in childhood.

Morbid Anatomy.—The changes vary according to the site of the inflammatory action. In *mononeuritis* the perineurium, or investing sheath of connective tissue which surrounds the entire nerve, is involved; hence such cases are described as examples of *perineuritis*. The outer sheath presents the usual characters associated with the inflammatory processes observed elsewhere—*i.e.* it is swollen or thickened, and red in colour. Its vessels are distended with blood, and small hæmorrhages into its connective tissue are frequently seen; the swelling and infiltration by leucocytes often forms a fusiform bulging at the affected spot in the course of the nerve. Where the action has been of long duration or of more intense nature, the changes characteristic of *interstitial* neuritis are observed. The exudation of leucocytes here, instead of being confined to the space between the perineurium and the nerve-fasciculi, invades the epineurium between the bundles of the nerve, and may even separate the individual fibres.

In typical cases of *multiple neuritis* the changes proceed still further, constituting the condition known as *parenchymatous* or *degenerative* neuritis, in which the fibres of the peripheral nerves break up and exhibit the characters known as the Wallerian degeneration, which have been always observed to follow after division of a nerve. In this form, which may be primary or may follow perineuritis or the interstitial variety, the axis-cylinder and the medullary substance, or myelin sheath, are broken up and disintegrated, and proliferation of the nuclei of the delicate primitive envelope, neurilemma, or sheath of Schwann, occurs. The degeneration diminishes as the affected fibres are traced upwards towards the cord, and becomes most intense towards the periphery. The broken-up

white substance appearing in flakes or droplets, together with the disintegrated axis-cylinders, is eventually absorbed. The end-plates degenerate simultaneously, the muscular fibres decrease in size, lose their striations, may become almost hyaline in appearance, and finally they present fibrous or cirrhotic changes from proliferation of their interstitial tissue. In the main, the changes in multiple neuritis are chiefly confined to the nerve-fibres, and in mononeuritis the connective tissue suffers most; in both conditions the spinal cord usually escapes entirely, though in some alcoholic cases its fibrous tissue is increased, or some chronic myelitis changes may be observed in disseminated areas.

Symptoms.—Perineuritis, or interstitial neuritis, which is the form usually seen when a single nerve is affected, does not give rise to constitutional symptoms. The chief feature is the severe pain felt in the region of distribution of the inflamed nerve; it is of a burning, boring character; it is always aggravated by movement, and is as a rule much more continuous than the intermittent pain of neuralgia. There is almost always marked tenderness to pressure over the inflamed nerve-trunk, which, if superficial, may be felt to be swollen or enlarged, and sometimes in this latter case slight redness and œdema of the overlying skin may be observed. Early in the progress of the disease there is paræsthesia and hyperæsthesia, and at a later stage anæsthesia. More or less paralysis and wasting of the muscles supervene, though at first there may be fibrillary twitches and startings, or involuntary contractions of the entire limb, as seen in severe neuritis of the sciatic nerve.

The electrical excitability is increased in the acute stage and diminished later on; similar changes may occur in the tendon-reflexes, which are usually entirely absent in advanced cases of neuritis; finally, the reaction of degeneration is observed in the muscles.

Trophic changes in the skin, which may become glossy or exhibit herpetic eruptions, occur; sometimes perforating ulcers or bed-sores form, and in severe cases joint-mischief may develop like that seen in some cases of tabes.

In the more chronic forms of interstitial and parenchymatous neuritis there is usually little or no evidence of swelling of the nerve, which is also much less tender to pressure than in the acute cases and the pain is generally of a milder type, though there is some hyperæsthesia, marked wasting, and paralysis of the muscles. These latter signs may be absent in mild cases of perineuritis which recover early.

In typical cases, where the traumatic cause is not a constantly acting etiological factor, recovery in a few weeks is the rule, as seen in the neuritis which follows contusions, dislocations, fractures, &c.

As the typical neuritis of isolated mixed nerves tends towards paralysis, the symptoms of these need not be here detailed, being briefly referred to in each case under 'Paralysis of Peripheral Nerves.'

MULTIPLE PERIPHERAL NEURITIS

Etiology and Pathology.—The chief points in the causation of this condition have been already mentioned upon page 1111. It is met with in its most typical form in alcoholics who freely consume beer and spirits; very rarely, if ever, is it seen in drinkers of pure wines. When occurring in those who drink beer it is believed by many only to appear when the beer has been contaminated with arsenic, as witnessed in the extensive epidemic in Manchester and elsewhere in 1900. Women suffer more frequently from alcoholic neuritis than men, and the majority of cases occur between thirty and forty.

Symptoms.—These are usually insidious, though occasionally the onset may be abrupt or acute, especially in those cases where the disease affects an alcoholic subject who is recovering from some form of infectious fever. The first symptom is often a prickling, tingling, or numb feeling in the tips of the fingers and toes; pain soon follows in the feet and hands, and often in the legs. It is of a dull gnawing character, is increased by movements and pressure, and is usually associated with hyperæsthesia, especially of the skin of the plantar surface of both feet, and with tenderness of the muscles, which is generally well marked in the calf of each leg if it be firmly squeezed by the hand.

Motor symptoms appear within a few weeks; the difficulty of walking, which was at first caused by the hyperæsthesia of the feet, is now increased, owing to actual weakness of the leg-muscles, and finally wasting and paralysis supervene. At this stage, in addition to the other symptoms of chronic alcoholism, the developed disease exhibits itself by the following changes in the motor and sensory systems. The tendon-reflexes are diminished and often entirely absent, though at the start they may have been exaggerated somewhat. The muscular weakness is bilateral and usually symmetrical, but generally more or less partial. Thus, even in the worst cases where the paralysis simulates typical paraplegia the affected muscles are not equally paralysed, some nerves suffering more severely than others. Thus the peroneal nerves are more deeply involved than the other branches of the sciatic, and consequently the muscles innervated by them usually first show paralysis, as seen in the characteristic 'dropped foot' and 'dropped toes,' the anterior tibial nerve, which is also a branch of the external popliteal, suffering along with the peroneal arising from the same trunk. When this latter nerve is less involved than the peroneal, inversion of the

foot is possible, though the feet and toes cannot be raised ; this is an important symptom showing the partial or selective nature of the paralytic poison.

The tibialis posticus, which is a continuation of the internal popliteal nerve, sometimes suffers early, as does also the crural. Finally all the leg-muscles may become paralysed, some power in the hip-muscles only remaining.

The legs waste, their muscles become atrophied and flaccid, though they may remain tender ; pressure elicits tenderness, and demonstrates the swelling of the nerves already mentioned. Contracture of the calf-muscles commonly supervenes upon paralysis of the ankle-flexors, and may prevent locomotion, even when considerable muscular power remains.

The degeneration of the muscles shows itself in altered excitability ; they respond less readily to galvanism and faradisation. Usually the reaction of degeneration is but a partial one ; it may be complete in long-standing cases, and sometimes in these the muscles and nerves finally totally fail in their response to the continuous and faradic currents.

Usually the muscles of the upper extremity escape ; when they suffer they present the same marked characteristics as seen in the legs—*i.e.* some of them may be deeply involved, whilst others supplied by the same nerve are able to exercise their usual function ; only the movements of the fingers and wrists are at first, or even throughout the case, affected, as in the lower extremity, where often only the toes and ankle-joints are crippled. The trunk-muscles escape, and the sphincters are unaffected.

Ataxia (pseudo-tabes) is sometimes present, as observed in the high-steppage gait ; and Oppenheim points out that in these cases spontaneous muscular twitchings are observable, of whose existence the patient is unconscious. Double facial paralysis has been occasionally observed.

In addition to the sensory disturbances, as pain, tingling, hyperæsthesia, and anæsthesia, already mentioned, there may be marked diminution of the muscular sense and delayed sensation. Very commonly these symptoms are not so obvious in the established disease as are the motor signs, but careful examination will usually detect various combinations of them, especially in the skin over the plantar surface of the feet, which is often hyperalgesic, though insensible to tactile impressions. These combinations, along with the muscular hyperalgesia, are highly characteristic of peripheral neuritis. The sensory changes are best marked in the distal portions of the limbs, and fade when traced upwards towards the spine ; they may be only demonstrable in the tips of the fingers and toes in mild cases. The skin-reflexes are diminished, and in severe cases may be absent along with the knee-jerks.

Vaso-motor and secretory phenomena may be evident, as seen in the congested and often œdematous state of the feet, and sometimes in the sweating of both feet and hands. Perforating ulcer and bed-sores rarely occur, but the skin may be glossy, and very occasionally joint-inflammation may develop. Mental symptoms are not rarely present, as described by Korsakoff, who defines the psychic features of the condition (alcoholic neuritis) as a polyneuritic psychosis in which mental confusion and impairment of the memory of recent events and delusions occur. Though this psychic disturbance is often well marked and presents a most striking feature in the disease, it may be sometimes seen in chronic alcoholics who present no evidence of peripheral neuritis, and it has been met with in the neuritis produced by other toxins and by arsenic.

Nystagmus is not unusual. Paralysis of eye-muscles sometimes occurs; the discs are generally normal, though a partial atrophy may occasionally be observed with a central scotoma for colours; the pupils as a rule are normal, and respond to light and accommodation. There may be tachycardia from involvement of the vagus, and paralysis of the phrenic may cause serious or fatal difficulty in respiration, the abdominal parietes below the thoracic margin being retracted in inspiration.

The predominance of the motor, sensory, or ataxic symptoms in individual cases has led to the division of alcoholic neuritis into different types, but in the great majority of cases the disease is met with in the mixed form, in which motor and sensory disturbances are always present together. Even in the best marked examples of the ataxic type, the muscular tenderness resulting from implication of the afferent muscle-nerves is usually well marked, though other sensory disturbances may be absent or slight; the incoördination of movement is obviously produced by the affection of these nerves. It should be remembered that this pseudo-tabes has been observed also in arsenical and other varieties of multiple neuritis.

The *course* of the malady varies; usually, however, the symptoms increase in severity for four, six, or eight weeks, and then in their fully developed state last for a similar period, after which they begin to decline, unless the toxic cause continue in operation. Recovery in all cases is slow and tedious. Occasionally the disease runs an acute course, beginning with fever, and it may terminate fatally within a few weeks.

Death may occur from heart-failure, paralysis of the diaphragm causing hypostatic pneumonia and bronchitis, or vagus implication may cause a fatal issue; sometimes hepatic

cirrhosis or pulmonary tuberculosis terminates the patient's existence.

Morbid Anatomy.—This has been described upon page 1112.

Diagnosis.—The chief point in the recognition of the disease is the combination of sensory and motor symptoms of a bilateral or symmetrical character; the sensory disturbances, as already described, showing themselves by the pain and tenderness in the skin, nerve-branches, and muscles, and the motor symptoms demonstrating their presence by the bilateral foot-drop and wrist-drop, with absence of knee-reflexes, and of sphincter-troubles.

Poliomyelitis anterior acuta in children is easily differentiated by the age of the patient, but when the affection occurs in adults the diagnosis may be difficult, and most authorities regard such cases as examples of multiple neuritis. The following are the chief differential points: in poliomyelitis the onset is sudden, and wasting of the muscles *rapidly* follows; the paralysis is not symmetrical, and it is not progressive; the pain is much slighter, the nerve-trunks and muscles are not markedly tender, and there is no anæsthesia. The electrical reactions, it should be remembered, may be similar in both diseases, but though the excitability to faradism may be entirely lost, whilst galvanism causes muscular contraction in each affection, the current necessary to produce this latter effect must be much stronger in multiple neuritis than in poliomyelitis of recent origin.

Locomotor ataxia presents considerable difficulty in its elimination, especially when it occurs in the type known as neuro-tabes, since here the chief symptoms are those of nerve-degeneration with the cord intact. In tabes the pain may be identical with that of neuritis, but the true lightning-pains are seldom, if ever, present in neuritis. The Argyll-Robertson pupil, atrophy of the disc, girdle-sensation, bladder-symptoms, the characteristic crises, and enlargements of the joints are not seen in neuritis. The steppage gait of neuritis is different from the typical ataxic walk, and usually the patient is able to stand with his eyes closed; foot- and wrist-drop seldom are present in tabes. The history of previous syphilis and the progress of the case assist in the diagnosis of ataxia, whilst the history of a recent toxic cause is usually obtainable in multiple neuritis.

Compression-myelitis affecting the lumbar region, or a transverse cord-lesion causing paraplegia and wasting of the leg-muscles, with loss of tendon-reflexes, is recognised by the implication of the sphincters and the marked tendency towards the formation of deep bed-sores; tenderness of the muscles is absent, but anæsthesia is present, which is, however, found to correspond in its horizontal distribution to the areas supplied by certain levels of the cord.

Acute ascending paralysis of Landry simulates acute rapid multiple neuritis closely, but the palsy in its ascent from the lower extremities involves the trunk-muscles before it affects the hands and wrists, whilst in neuritis the feet and hands are affected simultaneously or successively, the trunk being involved last; and in typical cases of Landry's paralysis there is no anæsthesia.

Rheumatism is simulated in many instances, and a considerable percentage of the cases do not present themselves for treatment till the established stage of the disease has been reached, because of the common mistake of regarding the sensory disturbances as rheumatic. The seat of the pain should be carefully located in all cases: in rheumatism it is in the joints, in neuritis it is in the muscles and along the nerve-trunks. It must not be forgotten that movement of a healthy joint may become painful owing to the implication of nerve trunks or branches in its vicinity. The tingling in the extremities and the anæsthesia are never symptoms of rheumatism.

Neuralgia is differentiated by its unilateral distribution, which easily prevents its being mistaken for multiple neuritis, but the diagnosis of the neuritis of a single nerve presents often great difficulty. As stated under 'Neuralgia,' the chief differential points are in neuritis a more spontaneous and continuous type of pain, with tenderness to pressure along the entire course of the inflamed nerve, which, when superficial, may be found to be swollen. There is generally some cutaneous anæsthesia with the hyperæsthesia; the characteristic circumscribed pressure-points seen in neuralgia are absent, and the supervention of paralytic and altered electrical excitability ultimately clear up the diagnosis.

The diagnosis of the different forms of multiple neuritis is as a rule easily accomplished by a scrutiny of the history and the discovery of the toxic cause, it being always kept in mind that very often a combination of two or more causes may be present. For the diagnosis of *beri-beri* see page 103.

Trichinosis has been mistaken for multiple neuritis, but the recognition of the characteristic nematode in the affected muscles is conclusive; the oedema, pain in moving the eyeballs, high fever, voice and respiratory troubles, should arouse suspicion of trichinæ at an early stage.

Prognosis.—In alcoholic multiple neuritis, and indeed in most forms of neuritis, the prognosis is good when the disease is seen in its early stages. Only in acute cases with elevated temperature, and in chronic neglected cases of long standing, is there any considerable danger to life from paralysis of respiration owing to intercostal or diaphragmatic incompetency. Evidence of heart, kidney, or liver degeneration, and of phthisis, deepens the gravity of the disease, even when the

motor symptoms may not be very marked. A very rapid weak pulse is a serious sign indicative of cardiac change or vagal involvement, or of both complications combined. Recovery is usually more rapid in the sensory form of peripheral neuritis, and when the symptoms are confined to the lower extremities.

The motor weakness usually shows signs of improvement within three or four months, but often six to twelve months may elapse before the extensors regain their power, and usually a longer period elapses before the wasting disappears in the affected muscles.

The degree of altered electrical irritability is an important point in the prognosis, the progress towards recovery being always very slow when this is well marked. An early return of faradic irritability in nerves and muscles is indicative of a satisfactory issue, provided always that the toxic cause is removed. The prognosis in lead and diphtheritic neuritis is referred to under their respective headings.

Treatment.—The first step in the management of any case of peripheral neuritis is the removal of the toxic cause of the disease. In the majority of all cases, this being alcohol or a combination of arsenic and alcohol, the measures referred to under 'Alcoholism' are indicated. Rest in bed is essential in all severe cases, with such precautions as are necessary to prevent the supervention of contractions or permanent deformities, owing to the tendency of the feet to drop and of the calves and hamstring muscles to contract.

Cotton-wool under oiled silk, warm fomentations, and warm, but not hot, baths, are useful for the alleviation of pain when not severe enough to call for morphia hypodermically.

Drugs are of secondary importance to the cessation of the poisoning and to rest, careful nursing, and judicious feeding. The best routine-drug is antipyrin in small doses, phenacetin, and the other analgesics and salicylates are also useful; it is a good plan to alternate them for short periods, and they may be employed with greater advantage by administering the liquid extract of coca in combination. Iodides are useful in the arsenical, rheumatic, and purely sensory forms. Aspirin often gives excellent results even when there is no evidence of a rheumatic origin of the neuritis.

Electricity should not be employed in the early stages, neither should massage nor strychnine hypodermically be used; but these three agents are the most valuable means at the disposal of the physician for aiding the nutrition of the muscles after paralysis has supervened. Faradisation of the affected limbs should be perseveringly tried, using large moistened electrodes and a current only strong enough to produce mild contractions of as large a number of muscular fibres as possible.

Swedish movements, Schott manipulations, and passive motion should be systematically practised.

Other forms of multiple neuritis may be briefly referred to ; those occurring in lead-poisoning, diphtheria, beri-beri, leprosy, malaria, diabetes, and influenza are mentioned or described under the respective headings of these diseases.

ARSENICAL NEURITIS

This sometimes follows recovery after acute poisoning by the drug ; very occasionally only is it produced by its medicinal administration in skin-diseases and in chorea. More commonly it is observed in operatives inhaling arsenical-laden dust in various occupations, or in persons living in rooms furnished by arsenical muslins or arsenical wall-papers. Of late years, however, by far the most common source of chronic arsenical poisoning has been traced to the drinking of beer contaminated by the presence of arsenic in the glucose employed in its manufacture. There cannot, however, be a doubt that the examples of multiple neuritis in the beer-poisoning epidemic were due to the combination of alcohol with arsenic. A corresponding amount of arsenic administered alone would probably have failed to produce symptoms of neuritis.

In uncomplicated arsenical neuritis the disease may appear abruptly, but much more frequently it is gradual in its onset, presenting symptoms and signs, in the main, almost identical with or closely resembling alcoholic neuritis. The following differences may, however, be observed : there are marked gastro-enteric symptoms usually in the cases following acute poisoning ; coryza and lacrymation, with suffusion of the conjunctiva, are more frequent in cases where the poison comes directly into contact with the respiratory tract. The pain, paralysis, hyperæsthesia, anæsthesia, and tenderness of the muscles, accompanied by wasting, are all present ; but the muscle-tenderness and pains are usually more marked than in alcoholic neuritis. An important distinction may often be made in the distribution of the paralysis, which is more frequently found affecting all the four extremities ; and the *flexor* muscles of the hands and feet are often paralysed along with the extensors—a condition but rarely met with in alcoholic cases. Ataxia is more common in arsenical neuritis, which may closely simulate genuine tabes, the reflexes being also absent ; there is considerable fibrillary twitching of the muscles. Skin-changes are more frequently observed, chief of which are greatly increased pigmentation or deep bronzing, hyperkeratosis of the soles and palms, desquamation, glossiness, pemphigus, herpes zoster, and erythema ; shedding of the hair is not uncommonly observed. The pathological anatomy is similar to that observed in the alcoholic form,

but there appears to be more liability to degenerative changes in the cord. The symptoms in the beer-poisoning epidemic, whilst mainly those of typical alcoholic neuritis, were combined with those of the skin-signs, notably keratosis, often associated with pigmentation resembling Addison's disease.

Diagnosis.—The investigation of the history and an examination of the patient's environment usually settle the diagnosis without difficulty, and arsenic is easily detected in the urine by Reinsch's test.

Prognosis.—The outlook is always good; death very rarely occurs if the condition be recognised early and further poisoning be prevented, though recovery may be very protracted. The muscles last affected first show signs of improvement, as seen in the diminution of their electrical degenerative changes.

Treatment.—This is practically identical with the management of alcoholic neuritis. Surgical methods may be demanded for the relief of contractures or deformities caused by the loss of the antagonism of the paralysed muscles.

MERCURIAL NEURITIS

This has been sometimes observed in syphilitic patients under prolonged courses of the metal, and it is doubtful whether the condition is to be attributed to the mercury or to syphilis, or to the combination of both poisons, or to their association with alcohol. When the old process of mirror-manufacture was in full operation, mercurial poisoning, associated with nerve-symptoms, was very commonly observed in the operatives, and it is still to be met with in thermometer-makers and workers in mercurial mines.

The chief nerve-sign is the characteristic *tremor*, which resembles that seen in paralysis agitans; but sensory symptoms are also common, as joint-pains and analgesia; mental or psychical disturbances are also often present. The results of experiments in animals show that degeneration of the medullary sheaths of the muscle-nerves can be produced by mercurial administration, but their axis-cylinders escape. Some are of opinion that the mercurial tremor is the result of alteration in the brain-substance and is independent of any change in the spinal cord. Oppenheim has observed that multiple neuritis occurring in syphilitic subjects is aggravated by mercurial treatment, and he thinks that there is no distinct evidence that pure mercurial neuritis ever occurs.

PUERPERAL NEURITIS

A mononeuritic and a polyneuritic form of the disease have been described as occurring in women during pregnancy or soon after delivery, the former affecting some of the nerves arising from the brachial plexus or the leg-nerves,

and the latter involving the peripheral nerves generally and sometimes those of cranial origin. In this multiple form the symptoms simulate those of alcoholic neuritis, but in the main they more closely resemble the clinical picture observed in post-diphtheritic and Landry's paralyses. Optic neuritis has been several times observed. The pathology of the condition is evidently that of puerperal sepsis, the anatomical changes being those of a peripheral parenchymatous neuritis, though minor changes in the cord have been noticed.

It must be remembered that instances of multiple neuritis are not rarely met with in which the toxic agent cannot be discovered; these may even appear in an epidemic form, like the beri-beri neuritis. Many of these so-called idiopathic cases are probably examples of post-diphtheritic or influenzal neuritis in which the symptoms of these primary diseases had been so slight as to escape notice.

NEUROMATA

New-growths or tumours of various kinds are found on nerve-trunks or their branches; thus, syphilitic or malignant neoplasms are not uncommon, though these are secondary to deposits in the tissues or organs in the vicinity of the affected nerves. According to some pathologists, a *true* neuroma—that is, a neoplasm made up of nerve-fibres or ganglionic cells—is not found in the body; if such exists, it is evidently of little clinical importance. The great bulk of all tumours arising in nerves are, strictly speaking, *false* neuromata—that is, they are neoplasms made up of proliferations of the fibrous-tissue elements in the perineurium or in the finer sheaths covering the individual bundles of the nerve-fasciculi. According to the modifications in the new connective-tissue growth, such false neuromata are described as belonging to the simple fibrous, myxomatous, sarcomatous, or lipomatous types of neoplasm, which in their growth only displace the nerve-fibres and nerve-cells. The so-called amputation-neuromata, which grow upon the divided centric ends of nerves, are the result of inflammatory changes in the axis-cylinders, and do not belong to the category of the neoplasms under consideration.

Neuromata are often encapsuled and solitary, especially when of the simple fibrous type, forming slowly growing, painful, but innocent, tumours, usually of small size, though occasionally they may assume very large dimensions. Of such are the small, very painful, tender, firm, subcutaneous *nodules* or *tubercula dolorosa* which are found growing upon the peripheral sensory nerves; occasionally they are solitary,

but more frequently they are multiple and found in very considerable numbers.

The ordinary fibromata, or neuro-fibromata, as they are called, may arise from any spinal nerve; not rarely they are found on the cauda equina, and sometimes on the nerve-roots within the vertebral canal. They may be limited in their distribution to one or more nerve-trunks or their branches, or they may be scattered in hundreds over the nerve-branchings throughout the body. When deeply situated they may produce no symptoms, save that the larger ones may cause disturbance by their bulk and pressure. They are of congenital origin; often a distinct hereditary history may be obtainable, so that the condition may exhibit the characters of a family-disease, sometimes, however, only developing after puberty. This feature is best seen in the *plexiform* neuro-mata, or so-called congenital elephantiasis, in which numerous soft and often pendulous tumours, consisting of thickenings on the tortuous interlacing cords or nerve-branches, are found limited to some region, as that of the trigeminal or side of the neck.

Another form of the disease is described as *diffused* or *generalised neuro-fibromatosis* by von Recklinghausen, which resembles the plexiform, but, unlike the latter, is not limited to the nerve-fibres in any particular region, though this generalised form may be sometimes associated with the plexiform condition confined to a localised area. Congenital nævi, various circumscribed pigmentations, and mental weakness or peculiarities are usually found in association with this variety of the disease.

The affection recognised as *molluscum fibrosum* is but a form of neuro-fibromatosis exhibiting itself *in the multiple but not in the diffused type*. (See under 'Molluscum Fibrosum,' p. 1049.)

The majority of all the forms of neuromatous growths may be met with devoid of any symptoms save those produced by their bulk. They are sometimes discovered accidentally, when they are liable to be mistaken for fatty tumours; they may remain latent throughout, and, unless they take on the sarcomatous type, they produce no constitutional disturbance. The isolated varieties may be excised; sometimes they can be enucleated from their capsules without injury to the nerve-trunk when operative interference is demanded by the presence of symptoms of tingling, anæsthesia, neuritis, or neuralgia occasionally arising from the pressure which they exercise. They seldom produce paralysis save when the cauda equina is the seat of their growth, in which case bladder-symptoms and paraplegia call for their removal. In the generalised variety little can be done for the relief of pain when this exists, and death from marasmus sometimes follows when the neuralgia

is severe. If removal is attempted in this form of the disease, the growths are very liable to return in an infiltrating sarcomatous type; but when only a limb is involved amputation may be permanently successful. The tubercula dolorosa have been relieved and sometimes caused to disappear by the application of strong continuous currents. Drugs, save for the relief of pain, are useless, but syphilitic nerve-thickenings may be removed by large doses of iodides.

NEUROSES

The term 'neurosis' is variously applied; generally it is used to designate the functional affections of the nervous system whose anatomical seat is unknown. Hence the neuroses are accepted as embracing a long list of diseases, like hysteria, neurasthenia, hypochondriasis, and the various psychoses or functional disorders affecting the mind. Under the term Spasmodic Neuroses, such affections as chorea, paralysis agitans, tetany, wry-neck, and various other forms of spasms are included. The Paroxysmal Neuroses are still considered as embracing such diseases as migraine, epilepsy, vertigo, eclampsia, &c. The Occupation Neuroses, or Professional Spasms, embrace those affections like writers' cramp, where spasms, are excited by attempts at performing some muscular movements necessary in the carrying out of certain occupations, as those of the telegraphist, piano-player, violinist, tailor, cobbler, cigar-roller, dancer, blacksmith, milker, watch-maker, &c. Under such terms as Gastric and Cardiac Neuroses the various forms of functional derangements of these organs are included.

Each diseased condition will be found described under its own name.

NIGHT-TERRORS

This is the title, along with that of *Pavor Nocturnus*, which has been given to the condition of terror observed in young children who upon partial waking (usually with a scream) from sound sleep appear to experience some distressing hallucination or vision. The symptoms closely resemble those seen in an attack of nightmare in the adult, and both conditions, as pointed out by Coutts, are constantly confounded with each other. This authority recognises that in nightmare the child dreams dreams, whilst in night-terrors there is always a vision of some frightful or distressing kind which seldom changes in the subsequent attacks. The child during a mild seizure often does not thoroughly wake up or become quite conscious of his environment; soon afterwards he falls

soundly asleep, and in the morning is usually unaware that anything unpleasant has transpired during the night. In severe attacks the intensity of the terror thoroughly awakes him, and he has a distinct recollection of his mental suffering in the morning.

Children between two or three and eight years of age are much more liable to attacks than older subjects. Guthrie maintains that no distinctions exist between night-terrors and nightmare; he recognises an idiopathic form as being of cerebral origin, and a symptomatic form due to reflex irritation.

There is nearly always a neurotic history in the idiopathic variety, and the child is generally apparently hypersensitive and often timorous in the dark. In many cases the effect of severe efforts in mastering school-lessons or the impressions produced by ghost-stories or practical joking are the obvious causes, especially in shy and precocious children.

In the reflex variety of night-terrors, nightmare of the adult is very closely simulated, if the two conditions be not identical. Intestinal worms, acute dyspepsia, delayed dentition, the presence of enlarged tonsils or adenoid growths, ear and eye troubles, are all capable of inducing attacks, which, unlike the seizures of true cerebral origin, may occur more than once during the night. Such attacks may occur in children up to the age of twelve, and are prone to recur during the progress of some febrile affection. Occasionally attacks may occur in the daytime, for which the term *day-mare* has been coined, and such seizures may be either of cerebral or reflex origin.

The diagnosis is obvious, but when convulsions have preceded the attacks there may be at first a difficulty in excluding brain-affections. The relation of night-terrors to epilepsy and other neuroses is a disputed point; probably their coincidence is caused by the same underlying inherited neurotic condition.

The prognosis is always good, but the neurotic condition so commonly met with in the cases of cerebral origin may blossom at a later stage into some psychical development, as somnambulism, insanity, migraine, hysteria, &c.

The treatment is obviously preventive in all cases where a removable cause can be discovered. The environment of the child should be closely examined, difficult school-lessons and excitements of all kinds must be strictly prohibited, and the nursery *régime* carefully regulated. Drugs are only indicated when the attacks recur with frequency; the best routine treatment is bromide of potassium in one full dose given a short time before bed-hour. It should always, however, be remembered that bromides in some patients are apt to cause vivid dreaming, which may intensify the tendency to nightmare in dyspeptic subjects.

NODDING SPASM. (See under SPASM.)**NYSTAGMUS**

This is the name given to the involuntary conjugate bilateral oscillating movements seen in the eyeballs, and caused by visual or muscular defects. The movements may be very rapid or slow, occurring in a horizontal, vertical, or rotatory direction. The most common form is that described as lateral nystagmus, in which the horizontal movements take place.

When the condition is trivial it may be only rendered observable when the eyeball is made to rotate extremely, inwards or outwards, upwards or downwards, or when the combined movements of circumduction are slowly effected. This form, which only occurs when weakened ocular muscles are put into strong action, is of little or no importance; it is described by McKisack as an intention-tremor; only the nystagmus which is characterised by more or less continuous oscillatory movements is of clinical import.

Though both globes are nearly always involved in the oscillations, the latter may be more obvious in one than in the other; the movements may be as slow as twenty and as rapid as two hundred per minute. They are always rapid in vertical and rotatory nystagmus, and may be either slow or rapid in the commoner lateral type; the range of the movements also vary very widely, but their rhythm is usually regular, and they cease during sleep.

Two classes of causes are recognised—viz. those which produce defects of vision and those which induce muscular disturbances, weakness, or paralysis. In the visual cases the nystagmus is usually the result of eye-disease occurring at a very early period of life, either in the form of a retrobulbar neuritis, choroido-retinitis, or an opacity caused by ulceration of the cornea or a congenital cataract; it is commonly seen also in albinos. Eye-troubles occurring in adult life seldom cause nystagmus, unless almost total blindness results; thus it is never produced by errors of refraction or cataracts, but it may follow optic atrophy.

The nystagmus which originates in muscular disturbances is of considerable interest and importance. It is most frequently of centric type, since it is practically always bilateral. Gowers believes that it is due to some derangement of the functions of the centres in the pons when the eye-muscles are the seat of spasm. The connection between the ocular, the motor, and the equilibrium centres is close,

and explains the nystagmus which sometimes accompanies vertigo in aural disease.

In *miner's* nystagmus, which has sometimes been described as a craft-palsy or occupation-neurosis, the oscillatory movements are generally regarded as evidence of fatigue or irritable weakness of the eye-muscles induced by ocular strain caused by the stooping or recumbent position assumed by the worker when he uses his pick, especially in dimly lighted galleries. Even in this form probably the influence of the semicircular canals is also at work, since the nystagmus often is only observable when the miner places his body in the recumbent or stooping posture, and in severe cases the objects looked at appear to dance before him, and giddiness or headache may be also present.

Diseases of the nervous system are often associated with nystagmus, the most important connections being those of disseminated cerebro-spinal sclerosis, *hereditary* ataxia, disseminated myelitis, and tumours of the cerebellum, in all of which conditions it may be most clearly marked whilst vision is perfect. It is also not infrequently seen as a symptom in tumours in various other situations, as in the pons, thalamus, and crus, in softenings, hæmorrhages, meningitis, sinus-thrombosis, hydrocephalus of the chronic type, and basilar inflammations.

OBESITY

This term, practically synonymous with corpulency, is applied to a condition characterised by an over-growth of fat in various regions of the body, especially in the subcutaneous tissue and within the abdomen.

Etiology.—Heredity plays an important part in obesity, many, but not necessarily all, of the members of a family exhibiting a tendency to the excessive development of adipose tissue, either in very early or late life. Age is an element in most cases, the condition showing itself most frequently after middle-age, and in women after the climacteric period; moreover, women, upon the whole, are more prone to obesity than men—a statement especially true of the Hebrew race. Sexual activity rather tends to counteract any tendency towards obesity, and the deposition of fat is markedly increased after removal of the testes and ovaries; a similar condition often supervenes upon amenorrhœa, and also after atrophy of the testes. Race is a powerful factor, Orientals after middle-life being especially liable. That this is not mainly a question of climate and indolence appears to the writer to be probable after observing many cases of obesity in the West, in the Red Indian squaws of Canada. In the Hottentot women the deposition of fat is often localised in the gluteal regions.

Diminished exercise and the indolent habits usually associated with the phlegmatic temperament are commonly observed factors in the production of obesity, and they are usually combined with an abnormal indulgence in sleep.

Food is a factor whose importance has been often as unduly exaggerated as it has been minimised. Many obese subjects are not given to over-eating; such are probably the sufferers from strong hereditary predisposition or from some diseased blood-condition, as is seen in many cases of chlorosis, anæmia, gout, and even in rickets. Much difference of opinion exists about the relative power of fats, carbohydrates, and proteids in causing an excessive deposition of body-fat. It is, however, certain that in many individuals excess of any form of food is sufficient to cause obesity, especially when associated with restricted exercise. The personal equation must never be lost sight of: some persons accumulate adipose tissue only from a carbohydrate-diet, others from a fat-diet; and it must also be remembered that proteids may become a source of fat. Carbohydrates, as a rule, are more potent agents in the production of obesity than the other classes of food; not however, because they are directly converted into fat, but owing to the rapidity and ease with which they are burned up in the system, they favour the formation of fat from the albuminoids, and at the same time also prevent the necessity for the combustion of the fatty ingredients in the diet. Alcohol in every form, especially when used as stout and beer, acts in the same manner, and by saving the combustion of fats and proteids aids in the deposition or accumulation of fat in the tissues. Alcohol also tends to produce fatty degeneration, which affection is to be distinguished from mere obesity. In the former condition the protoplasm of the body-cells is converted into fat; in the latter the fat is merely deposited in excess in those tissues and cells which normally contain it.

It will thus be seen that as regards the etiology of obesity two well-marked types may be demonstrated—viz. the one of hereditary origin, in which the adipose condition is maintained upon an apparently moderate diet of almost any kind of food; whilst in the acquired type, over-eating, insufficient physical and mental exercise, alcohol, and the reduction of the metabolic processes which occurs after middle-life, chiefly in women, are the main factors. When both classes of factor are combined, the most aggravated examples of obesity are the result. About as many cases of the acquired form are met with in practice as of the hereditary type.

The symptoms and signs of obesity are too obvious to require description. They are those of the general plethoric state. When the condition persists, serious changes may result; thus the heart, which at first must necessarily undergo some degree of hypertrophy, tends at a later stage to dilate, and

symptoms of failing compensation may supervene. Arterio-sclerosis is often met with : in the writer's experience this is much more likely to supervene in the acquired variety of obesity. Owing to the deposit of fat in the liver and upon the surface of the heart, these organs may be considerably impeded in their functions, and the liability to true fatty degeneration of all the organs must not be lost sight of. Obese subjects are prone to glycosuria, which, after running a chronic course, may end in diabetes. Gouty symptoms are often associated with the appearance of the sugar.

Obese patients are, upon the whole, more prone to succumb to acute illnesses of various kinds, and the condition does not lead to longevity, as proved by the statistics of insurance societies. Surgical operations are very badly borne by such subjects, and the risks of operations on the bladder, kidney, and on umbilical hernia, and especially of the operation necessary in gall-stone obstruction (all of which may be necessitated by the changes indirectly due to the obesity), are exceedingly high. The excessive death-rate in such cases is mainly due to collapse from heart-failure. Apoplexy, diabetic coma, angina, cardiac syncope, and uræmia are common causes of death in advanced obesity.

The class of case characterised by pallor and anæmia is met with most frequently in girls who suffer from chlorosis or amenorrhœa and in women subject to menorrhagia, or in men affected with hæmorrhoidal or other bleedings. The main symptoms in these are palpitation, breathlessness on exertion, more or less anasarca, a quick low tension and feeble pulse, great languor (both physical and mental), and a dangerous tendency to syncope. The tissues are very soft and flabby, the appetite is poor, and there is usually a marked tendency to sleep. Such cases were formerly included under the category of 'serous plethora,' owing to the obvious hydræmic condition of the blood and its deficiency in hæmoglobin.

ADIPOSIS DOLOROSA

This is a form of obesity in which the fatty deposit, instead of being uniformly laid down throughout the subcutaneous tissues or within the abdominal cavity, is found to be limited to certain areas on the limbs or trunk. It was described by Dercum, and is sometimes called by his name; the condition is met with in middle-aged women. The soft, nodular, somewhat diffuse or lumpy, fatty deposits are to be distinguished from ordinary lipomata and from the plaques seen in myxœdema. The distinguishing feature in all cases of this rare disease is severe burning pain of the character of an acute neuritis, which is often also associated with some paræsthesia, hyperæsthesia, or anæsthesia of the skin areas

in the neighbourhood of the deposits. The thyroid gland having been found extensively diseased at the *post-mortem* examinations of two fatal cases suggested an explanation of the pathology of the disease being akin to that of myxœdema, but the signs of this affection were not present. It is, however, a significant fact that the symptoms have been markedly relieved by the use of thyroid feeding.

Treatment.—In the author's 'Dictionary of Treatment' (4th ed.), pp. 618-625, the details of the various methods of dealing with obesity and the formulæ of several of the dietaries commonly recommended are given. Only the general plan of treatment need be here briefly referred to. Preventive measures are indicated in all cases when the hereditary tendency is well marked. These are dictated upon ordinarily recognised principles. A carefully regulated diet is indicated, in which carbohydrates are very sparingly used and fats only permitted in moderate amounts, starch, sugar, and alcoholic beverages being especially restricted, and the gross intake of food kept as far as possible within the necessities of growth and development. Of even more importance is the regulation of the individual's habits as regards outdoor life and active exercises in the open air, especially as the hereditary tendency to obesity is so often associated with the phlegmatic temperament. Sea-bathing, swimming, and games necessitating active muscular movements are called for in young subjects. In persons exhibiting hereditary tendency to obesity after middle-life the intake of food can be diminished with greater safety and the supply of fluids restricted, as much less is required to meet the demands of the system at this period; but the muscular exercises may with advantage be sought in regular systematic walking, cycling, and in such games as golf.

In the established condition the same principles must be observed in the restriction and selection of the different foods, but due care is necessary in all cases at the start to determine the presence of any structural cardiac, renal, or other lesion. When such exists, the diminution in the total amount of food administered must be gradually and carefully regulated, and in all such cases the patient should give himself up to hospital treatment or routine supervision by the physician in his own home; by this plan a much more marked reduction in the total quantity of food taken can be effected with greater safety.

The usual calculation of the amount of food necessary to maintain an averagely active adult in health is roughly about 3 oz. fat, 4 oz. proteids, 12 to 14 oz. carbohydrates, and 100 oz. water. The amounts and proportions of these ingredients vary greatly in the different dietaries used in the treatment of obesity; no hard-and-fast rule can be applied till the

powers of the individual in each case are tested by careful observations made upon the reduction of body-weight, the activity of the digestion, and the output of urea.

After the total quantity of food in a diet consisting of the above-mentioned classes of ingredients has been diminished for a time to the minimum normal amount, the proportions of each class of food are to be adjusted to the necessities of each case. Carbohydrates should be restricted first, afterwards the fats, and finally the fluids, always remembering that not only is the abnormal amount of fat in the body to be removed, but its subsequent accumulation prevented. Oertel's daily diet consists of about 6 oz. proteids, $1\frac{1}{2}$ oz. fats, and about 3 oz. carbohydrates, with a restriction of fluids to less than 2 pints, graduated exercises being constantly performed throughout the treatment.

Some authorities recommend a *dry* diet as of prime importance; others consider that abundance of hot water, with an almost unlimited amount of beef, is better; and Yeo recommends a restricted amount of pure milk, whilst the patient is kept in bed for long periods. He instances Debové's case, in which a reduction of almost four stones was accomplished by this plan during twelve months.

The basis of all treatments has for its object such a reduction in the total quantity of food administered (especially in the carbohydrates) as will ensure the patient burning up his superabundant supply of adipose tissue. After this has been accomplished its re-accumulation is to be prevented by regulated exercises and such changes in his permanent dietary as can safely be made without reducing further his body-weight and strength.

Drugs are as a rule contra-indicated in all methods of treating obesity; the supposed success following the use of them is to be explained by the restriction of diet and the increased exercises which form a part of all the successful plans. Thyroid extract undoubtedly possesses the power of reducing body-weight, but its use for this purpose, if long continued, is fraught with considerable danger. It has, however, proved of value in the treatment of *adiposis dolorosa*.

OBSTRUCTION OF THE BOWEL.

(See under **INTESTINAL OBSTRUCTION.**)

OCCUPATION-NEUROSES.

(See under **CRAMP**, p. 248; and under **NEUROSES.**)

OEDEMA. (See under **DROPSY**, p. 315.)

ŒSOPHAGUS, DISEASES OF

The gullet is affected by inflammatory and malignant diseases and strictures, as well as being liable to suffer from pressure from without.

ŒSOPHAGITIS

Etiology.—This portion of the alimentary tube is occasionally the seat of an *acute inflammation* from the extension of a catarrhal or aphthous inflammatory process in the pharynx. Acute œsophagitis has also been observed, though rarely, to occur in the eruptive fevers, especially typhoid, scarlatina, and small-pox; not infrequently it may be the result of the diphtheritic process. Malignant growths or infiltrations of the tube are also generally associated with more or less chronic catarrhal inflammation.

The most common cause, however, of acute œsophagitis is trauma, the result of the passage of bougies or the swallowing of hard angular bodies, as pieces of bone, plates with artificial teeth attached, and corrosive liquids. The latter class of case is of the greatest clinical importance on account of the immediate danger to life and the liability of its being ultimately followed by structural narrowing of the passage.

Symptoms.—The symptoms following the administration of corrosive or caustic liquids, like strong vitriol, nitric acid, or ammonia, are those of shock accompanied by severe pain in deglutition and continuous dull pain behind the sternum, followed after a time by symptoms of serious gastric inflammation. The immediate result is the destruction of the mucous membrane at various sites, and the formation of sloughs, followed by acute œdematous inflammation of the coats of the gullet, which may in some cases extend to the mediastinal tissues and organs. Upon the separation of the sloughs, should the patient survive the initial shock, there may be severe fatal hæmorrhage or perforation.

Treatment.—The treatment should be directed to the speedy neutralisation of the corrosive liquid by alkaline or mild acid drinks, followed by demulcent mucilaginous or oleaginous draughts or egg-albumin. It is needless to remark that the stomach-tube should not be resorted to; unless in very exceptional cases even the soft rubber tube is not permissible. Food should consist of iced milk or egg-albumin, and where dysphagia is severe resort to rectal feeding is imperative.

CANCER OF THE ŒSOPHAGUS

Etiology and Pathology.—The growth is commonly of the epithelial type, composed mainly of squamous celled elements; starting in the mucous membrane, it is liable to

end in ulceration and infiltration of all the coats of the tube, may ultimately invade neighbouring organs, especially the glands in the posterior mediastinum, and often causes secondary deposits in distant organs. Cancer of the œsophagus is more common in men, and, like the disease appearing in other organs, it exhibits the usual characteristic etiological features as regards age, heredity, &c. Any portion of the tube may be the site of the disease; usually the narrowest parts are most frequently affected—viz. about the middle third and near to its entrance into the stomach.

Symptoms.—Dysphagia is commonly the first indication of the disease; it is observed in the early stage on some attempt to swallow a larger morsel than usual of solid food, whilst the swallowing of liquids is still unattended by difficulty. Gradually, as the obstruction becomes more complete, all food passes downwards under considerable difficulty, and finally the symptoms of total obstruction appear. With the act of swallowing in the early stages there may be severe pain; but often this is absent, and as a rule it does not appear till ulceration takes place, or until some inflammatory action is excited in the mucous membrane, which causes reflex spasm. Occasionally a dull constant pain is complained of, independent of the suffering caused by attempted deglutition.

A characteristic sign of the disease is present, usually before the obstruction is complete, and it is especially well marked when the growth is low down. The gullet dilates, and its walls become hypertrophied above the obstruction; in the pouch so formed the food is retained for some minutes, to be afterwards regurgitated, sometimes being mixed or streaked with blood, and accompanied by the regurgitation of muchropy mucus. The writer has been struck by the frequency with which patients in the early stage of the disease describe their symptoms as being due to 'something coming up from the stomach which meets the food and prevents its getting down'—a symptom or sensation which may be caused by reversed peristalsis in the part below the disease.

During the progress of the narrowing, emaciation rapidly sets in, and soon becomes extreme, owing to the starvation being exaggerated by the usual cancerous wasting. Occasionally a lobe of the obstructing tumour disintegrates and causes a temporary relief by partial removal of the blocking, and such fragments are valuable diagnostic aids when found in the regurgitated food.

Sometimes perforation occurs, and rapidly fatal septic inflammation is started in the lungs, mediastinum, pericardium, or pleura. The liability to extensive and rapidly fatal hæmorrhage should always be kept in mind; the writer can recall the tragic case of a patient sitting at the fire in his hospital ward awaiting the passage of a bougie by a surgeon, for the

introduction of a Symond's tube, when, without any warning of previous hæmorrhage, a column of blood was forcibly ejected from the open mouth, soon followed by the copious discharge of bright arterial blood from the bowel, rapid shock and death following within a few minutes. The *post-mortem* revealed perforation of the aorta, which would have been certainly precipitated by the passage of a sound, had this been attempted at a previous period; fortunately, delay had been caused in procuring the tube. Perforation of the mediastinum and pleura by pointed or tapered gum-elastic bougies sometimes occurs in the hands of skilful surgeons during the examination of malignant strictures.

When the growth is situated in the upper part of the gullet enlargement of the cervical glands may be evident, and sometimes laryngeal symptoms are caused by involvement of the recurrent nerves, or by direct implication of the cords or cartilages.

Diagnosis.—There is usually little difficulty in determining the nature of the disease, owing to the extreme emaciation and rapidly progressive cachexia, which of themselves usually exclude aneurismal growths. The history of cicatricial narrowings always reveals traumatic causes, and the cervical glands are never enlarged. The site of the obstruction is demonstrated by the careful introduction of an olivary bougie; in disease of the cardiac end the block is found at about fourteen inches measured from the incisor teeth.

Prognosis.—This is always, it is needless to state, fatal, death usually occurring from progressive asthenia if not precipitated by some complication caused by perforation.

Treatment.—Dilatation of the narrowing by graduated bougies is never permissible. The best routine treatment is the introduction of a Symond's short gum-elastic or rubber tube into the narrowed portion; the tube is kept from falling downwards into the stomach by its upper funnel-shaped extremity, which rests in the dilated part of the gullet above the stricture, whilst its thinner lower extremity occupies the narrowed area.

Usually, immediately after the tube has been placed *in situ* the patient is able to drink freely and swallow pultaceous foods. Gastrostomy and œsophagostomy are seldom indicated.

CICATRICAL NARROWING OF THE ŒSOPHAGUS

As already stated, this follows the sloughing and ulceration produced by corrosive poisoning, scalds, injury by dental plates, &c.; occasionally it may be the result of syphilitic ulceration, and has been met with as a congenital condition.

The condition often yields to cautious dilatation by tapered gum-elastic bougies. Almost thirty years ago the writer successfully treated a case of extremely narrow stricture by

the introduction of fresh sea-tangle formed into a long tapered bougie and partially dried. After a moderate dilatation by bougies, a Symond's tube may be used with great advantage to complete the widening. Surgical procedures of a more serious kind may be demanded in impassable strictures, as œsophagostomy, or the making of an opening into the stomach for feeding purposes. Various sounds have been introduced by Senator, Alessandri, and others, by means of which the stricture can be stretched or the manœuvre of divulsion effected.

The complete obstruction of the gullet caused by the pressure of aneurismal and other tumours can only be relieved by an opening made into the stomach for feeding purposes in order to prevent death from starvation.

DILATATION OF THE ŒSOPHAGUS

As already mentioned, this is always present immediately above the stenosed ring. In cases of cicatricial stricture low down in the tube, the widening of the gullet may affect its entire length; it is doubtful if general dilatation ever supervenes upon spasm of the cardiac end of the œsophagus, or even upon malignant disease in the region. Mikulicz has reported cases of simple spasm where there was a marked dilatation of the gullet forming a pear-shaped sac with the small end upwards; the contents of this sac could be syphoned off without entering the stomach or disturbing its contents. A general widening or dilatation has been occasionally recorded in connection with congenital narrowing of the cardiac end of the gullet or stomach. Such cases fall entirely within the province of surgery.

DIVERTICULA or ŒSOPHAGEAL POUCHES

These are met with in two forms. When they occur high up they are usually the result of pressure caused from within by the distension produced by the food in deglutition acting upon a congenitally weak region on the posterior wall of the gullet close to its connection with the pharynx. The primary origin of such diverticula is probably due to abnormal persistence or irregularity in the position of a visceral cleft. They ultimately consist of a pouch of mucous membrane alone, the muscular tissue having entirely disappeared, and the food, accumulating in the thin distended sac, presses upon the gullet, causing dysphagia and efforts at regurgitation of the retained decomposing contents, or it may cause pressure upon the larynx or other structure, and may be visible and palpable in the neck as a soft tumour. Such pouches have been successfully removed by the surgeon; if let alone they tend to enlarge and finally perforate.

The diverticula which occur lower down have a different origin. They are situated usually at about the level of the tracheal bifurcation, where some old adhesive inflammatory process, arising in the mediastinal glands, has led to the adhesion of a portion of the gullet-wall to the inflamed adenoid capsules. The traction or dragging on the wall of the œsophagus, which occurs as the cicatricial tissue gradually contracts, produces pouching. Like the pressure-diverticula, these so-called traction-pouches are found eventually to become sacs consisting of mucous membrane only; they are prone to rupture or ulcerate into the aorta or pericardium. The pouches can be located by filling the gullet with bismuth emulsion and using the X-rays; by this method they can be differentiated from dilatations caused by spasm or stricture.

FOREIGN BODIES IN THE ŒSOPHAGUS

This condition is chiefly of surgical interest, and in many cases the foreign body consists of dental plates carrying false teeth; coins are common objects in this situation, and rapidly bolted masses of solid hard food may also become firmly impacted. Where the foreign body is of small dimensions and free from angular projections, if it cannot be extracted through the mouth or ejected by vomiting, an attempt may be cautiously made to force it downwards into the stomach. In all cases of serious impaction a speedy attempt should be made at its removal by opening the gullet in the neck or thoracic region, or if the lodgment be at the cardiac end of the tube the stomach should be opened. Impacted dental plates, if allowed to remain, are prone to cause ulceration, perforation, or fatal hæmorrhages. Radiography and auscultation are valuable methods for locating the foreign body when the passage of a sound is difficult or contra-indicated.

RUPTURE OF THE ŒSOPHAGUS

This is by no means a very rare accident where the gullet has been the seat of malignant ulceration, or where extensive pouching has occurred. Rupture has been known to occur in gourmands after a full meal of solid food, the healthy tube being torn in the act of violent vomiting even as low down as close to its entrance into the stomach.

SPASM or ŒSOPHAGISMUS

Etiology.—This condition, apart from those cases in which it may be a reflex manifestation caused by ulceration or obstruction, is seen in young neurotic subjects. In hysterical females and in hypochondriacal males it is not rare and is then found independently of any organic lesion. In one case recently under the writer's notice, typical attacks of spasm occurred for years in an elderly neurotic Hebrew woman,

extending over several days at a time, each seizure being brought on by any attempt at swallowing. The spasms would suddenly cease as abruptly as they commenced, and in the intervals swallowing was normal.

Symptoms.—These may commence suddenly with attempts at vomiting; more frequently there is not any retching, but regurgitation of food suddenly occurs during a meal, solids and liquids being brought back into the mouth. Usually there is a sense of constriction or pain, and emotional manifestations may accompany or precede the attack.

The seat of the spasm may be in the muscular coat at the upper end of the tube, but more frequently, as judged by the nature of the regurgitant act, it may be situated low down; hence it is usually described under the title of cardio-spasm of the gullet. The diagnosis is evident from the history of the abruptness and paroxysmal characters of the attack, and it may be sometimes demonstrated with accuracy by the easy passage of a full-sized bougie. This procedure is also the best method of treating the condition, which usually, for a time at least, ceases after the passage of the instrument. Where, however, considerable dilatation of the œsophagus has supervened in long-standing spasm at the cardiac end of the gullet, as in Mikulicz's cases, this authority recommends the manual dilatation of the narrowed part from below after making an opening into the stomach.

The opposite condition of *paralysis* of the œsophagus is very occasionally met with in hysterical patients, and is speedily removed by the œsophageal bougie. More commonly the dysphagia is either a part of the clinical picture of bulbar paralysis or a sequela of diphtheritic neuritis.

OLFACTORY-NERVE DISEASE

The only symptom produced by the arrest of function of the first cranial nerve is the loss of the sense of smell, or *anosmia* as it is called.

Etiology and Pathology.—Anosmia may be the result of injuries to the base of the skull and blows or falls on the back of the head. Inflammation in the anterior fossa involving the membranes or bones, especially if of syphilitic origin, and the pressure of tumours, may destroy the nerve in any part of its course, either in the bulbar portion or the olfactory tract.

Anosmia is, however, much more frequently the result of a diseased condition of the mucous membrane of the nosé which destroys the peripheral ends of the nerve. It is not a rare symptom of nasal polypi, and is found in disease of the fifth nerve; it is often observed in ataxia, where it is supposed to be due to degeneration of the nerve. With the

loss of smell there is loss of power in distinguishing the flavours of substances, though the sense of taste is not impaired; but in diseased states of the mucous membrane, if not severe, the ability to distinguish flavours may remain to some extent. Occasionally anosmia is the result of developmental anomalies, in which case it will be present from the time of birth, and sometimes it gradually supervenes in the senile period from wasting of the bulbs. Rarely it is associated with deep brain-disease, as when the posterior part of the internal capsule has been injured.

Anosmia is often observed in the hysterical condition, in which case it is usually transient and associated with hemianæsthesia. By far the most common cause in the experience of the writer is influenza, in which disease it may probably be part of a peripheral neuritis; and in many of these cases the sense of smell never returns, or it remains permanently impaired. Exposure to strong odours has been known to cause permanent loss of smell by over-stimulation of the nerve. The diagnosis should be made by testing the patient with such odorous substances as otto of roses, musk, &c., not by the use of volatile pungent bodies like ammonia or strong acids.

Prognosis.—This is unfavourable as regards recovery of the sense of smell when the condition is due to disease of the nerve, but a return to the normal is likely to occur in cases of disease of the nasal membrane which are curable.

The treatment consists in the local application of a weak solution of strychnine and the use of a feeble faradic current.

Hyperosmia, or increased sensitiveness in smell, is chiefly found in hysteria, and has been noticed in rare cases of total blindness occurring early in life.

Parosmia, subjective sensations or perversion of the sense of smell, may be produced by irritation of the nerve or its centre. Such olfactory hallucination is observed in some forms of epileptic aura and was once observed in a case of neurasthenia by the writer, where it remained for several years, but finally disappeared.

OPHTHALMOPLEGIA

This term may be taken as practically synonymous with *nuclear ocular paralysis*. It is a bilateral paralysis of the external or internal muscles of the eye, or of both sets of muscles combined, resulting from changes in the nuclei from which their nerves arise. The condition rarely exists as an independent disease, being usually associated with other diseases of the nervous system, as bulbar paralysis, tabes, disseminated sclerosis, and general paralysis of the insane. In the *external* form of ophthalmoplegia, as its name implies,

the muscles which move the eyeballs are involved, and usually also the levator palpebræ superioris; whilst in the *internal* variety the iris and ciliary muscle suffer, causing dilatation of the pupil and loss of accommodation.

The disease may be congenital, arising from developmental anomalies in the nuclear area, in which case the levator palpebræ usually suffers most along with the abducens, the iris generally escaping, even when the ophthalmoplegia is otherwise complete; occasionally there is some weakness observed in the muscles supplied by the seventh nerve.

Ophthalmoplegia is either *sudden*, *acute*, or *chronic*. In the *sudden* form of onset paralysis of the ocular muscles may be more or less complete in a few minutes from traumatic hæmorrhages into the central ventricles following blows; it may be the result of hæmorrhagic encephalitis, or of embolism of a basilar branch.

In the *acute* form the symptoms require some days for their full development. The etiology of this variety is believed to be an acute toxæmia, since the condition is usually observed in alcoholism, ptomaine or septic poisoning. The symptoms are those of irregular or asymmetrical paralysis of one or more of the external muscles in each eye, the internal muscles usually escaping. In a small group of cases commonly but inaccurately catalogued under acute ophthalmoplegia, the paralysis is the result of a peripheral neuritis, as sometimes seen in alcoholic, post-diphtheritic, and post-influenzal neuritis. Such cases may usually be differentiated from nuclear disease by the presence of the involvement of other nerves at the same time.

A transient form of the disease has been observed several times which comes on acutely and passes completely away in a few days or weeks.

The prognosis depends in all cases upon the cause of the ophthalmoplegia, being gravest where the paralysis shows signs of progressively involving all the muscles, as is witnessed in some alcoholic cases and in those examples of acute ascending polio-encephalitis inferior associated with bulbar paralysis.

Chronic ophthalmoplegia is much more common than the acute and sudden forms.

Etiology and Pathology.—This chronic type is progressive, being the result of a degenerative or chronic inflammatory process involving the ganglion-cells in the floor of the third ventricle and aqueduct of Sylvius, causing destruction of the ocular nuclei, the process being identical with the changes observed in the grey matter of the cord in progressive muscular atrophy. Ultimately the entire nerve-segment degenerates, its root, its periphery, and the muscles supplied by it undergo wasting.

The cause of the degenerative process is probably the result

of a chronic toxæmia, as evidenced by the association of the condition with the nerve-diseases following syphilis. Its relations with general paralysis of the insane, multiple sclerosis, progressive muscular atrophy, and chronic bulbar paralysis may also be accepted in support of this theory.

Symptoms.—The condition usually appears first as a ptosis or diplopia in adults or subjects at middle-life, more commonly males. After a variable period of months or years, most of the external ocular muscles on both sides are found involved, all the movements of the eyeballs being restricted or lost, in advanced cases giving to the patient a characteristic fixed staring expression. The paralytic phenomena are, however, often irregular and asymmetrical, the movements of one eye being often more affected than its fellow, and all sorts of varieties and combinations may be observed in this respect. The internal muscles may entirely escape in some cases—a point which is supposed to be diagnostic of the nuclear origin of the disease, since in bilateral disease of the nerve-trunks the sphincter iridis and the accommodation are always involved. More frequently, however, the internal muscles suffer to some extent, and when completely involved along with the externals the condition is known as ‘mixed or *total ophthalmoplegia*.’

The levator palpebræ escapes more frequently than the other muscles, and seldom is ptosis complete, even in advanced cases. Occasionally there is evidence of paresis of the orbicularis palpebrarum. Optic-disc atrophy is common, as might be anticipated from the frequent association of the condition with tabes, and often the symptoms and signs of the other associated nerve-diseases already mentioned are also present from the start.

The ophthalmoplegia may cease to progress at any stage; more usually it proceeds to an advanced degree before remaining stationary. Its duration and prognosis may be said to correspond with those of the primary nerve-disease; of which it is but a part. The condition may precede the symptoms of the more general nerve-malady, but ultimately, in the great majority of all cases, tabes, paralytic dementia, or other nerve-affection supervenes.

It must be remembered that the disappearance of diplopia is no indication of permanent improvement, as the double vision is usually only present in the early stages; and this is often noticeable also in the early stages of ataxia in cases where ophthalmoplegia does not supervene.

Treatment.—This will consist of the administration of remedies indicated in the management of the primary condition, and, since it is so frequently due to syphilis, mercury and iodides may be expected to give the best results. In the acute toxic cases the indications are equally clear. Attempts may be made to minimise the weakness of the muscles by

hypodermics of strychnine and by electricity ; diplopia may be relieved by prisms, and occasionally antagonistic contracture of muscles may be remedied by tenotomy.

OPTIC-NERVE DISEASE

To the physician affections of the optic nerve within the eye and behind it are chiefly of importance because of the light which they shed upon the diagnosis of many obscure nerve-diseases. It is in this connection that the following brief description is provided.

OPTIC NEURITIS or PAPILLITIS

is the term employed to designate inflammation of the terminal portion of the optic nerve (the optic disc) in the fundus of the eye.

Signs and Symptoms.—As seen by the aid of the ophthalmoscope, the first signs are those of increased vascularity and some blurring of the circumference of the disc. The natural cup-shaped central depression becomes more or less obliterated as the swelling of the disc increases and causes its prominence to be more evident, and its diameter enlarged sometimes to double or treble its normal dimensions, whilst its pink colour deepens to red or purplish-red and presents a striated appearance. At this stage small hæmorrhages are commonly seen in the swollen papilla about its margin ; though the arteries are but slightly altered the veins are dilated and tortuous, especially where they spread out on the retinal surface ; they may be entirely obscured in the disc itself owing to the effusion. The maximum degree of swelling may be reached in acute cases in a few weeks ; more frequently it is observable only after several months. The neighbouring zone of the retina participates in the inflammatory swelling when this has been intense, so that a true neuroretinitis is observed.

After a variable period, in mild cases the disc gradually assumes its normal colour and dimensions, the dilated veins lose their tortuosity, and complete resolution takes place. Where the inflammation has been intense and rapid the new elements develop into cicatricial tissue, which shrinks as the effused lymph becomes absorbed, leaving a white and atrophied disc and usually some degenerative changes in the neighbouring retina showing as white specks.

Vision may be entirely normal in even moderately severe optic neuritis, but often great impairment of sight follows, and in the rapid and intense forms, sometimes spoken of as *choked disc*, total blindness may result.

Etiology and Pathology.—The pathology of optic neuritis is that of an acute inflammation of the disc or papilla. Some authorities think that where the swelling is great the condition may be explained by the increased pressure of the cerebro-spinal fluid along the optic-nerve sheath in diseases which raise the tension of the fluid within the cranial cavity, and this theory probably gave origin to the term ‘choked disc.’ The majority of observers, however, maintain that the choked condition of the disc is not the result of any mechanical strangulation, but is to be regarded as evidence of a true inflammation of the nerve.

Optic neuritis is very frequently met with in cerebral tumour, and its presence is of the greatest importance in diagnosis ; both discs are usually affected.

Internal hydrocephalus, sinus-thrombosis, cerebral abscess, meningitis, especially of the basilar type, encephalitis, embolic softening arising in ulcerative endocarditis, and other brain-conditions have sometimes been found in association with optic neuritis. These causal factors are found less frequently than cerebral tumour, which is a very common cause of the condition.

Optic neuritis is found associated with diseases other than cerebral ; thus it has been observed in its typical state in anæmia and chlorosis, lead-poisoning, Bright’s disease, and also as a sequela or complication in the acute infectious fevers.

When the condition exists only on one side it will generally be found that the primary cause is situated in the orbit.

Retrobulbar neuritis is the condition in which there is inflammation of the optic nerve behind the eyeball ; the disc generally in these cases shows no sign of inflammatory action, but atrophy, usually to a partial extent, follows, as seen by pallor of its temporal half. This form of atrophy is known as ‘secondary,’ in contradistinction to the *consecutive* atrophy which follows severe inflammation of the disc, or papillitis. Retrobulbar neuritis in the great majority of cases is of toxic origin, and the various forms of toxic amblyopia are produced by it, as are seen in alcohol, tobacco, lead, salicylate, and quinine poisoning. Diminution in the acuity of vision and contraction of the field are the marked features of the condition. There is a central scotoma for red and green or a central loss of vision from *axial* neuritis, which is not uncommon in diabetes and sometimes in tobacco-amblyopia where the inflammation or degeneration affects the central fasciculus of the nerve. The toxic amblyopias and amauroses are often classed with the hysterical and uræmic form as functional affections supposed to be due to disturbances causing arrest of function in the cortical cells of the visual centres.

ATROPHY OF THE OPTIC NERVE

Primary atrophy is almost always seen to affect both eyes. It is recognised by the grey pallor of the disc, and is seen in its most typical states in tabes, also in general paralysis of the insane, and in a more or less incomplete form in disseminated sclerosis, in all of which affections it is not preceded by papillitis. Primary atrophy is sometimes seen in a congenital or hereditary form appearing in young male subjects.

It is to be carefully differentiated from the atrophy which is consecutive to acute inflammation of the disc caused by cerebral tumours and from the secondary atrophy of retro-bulbar neuritis, which latter toxic form may be distinguished from the other atrophies by the fact that the loss of vision precedes the pallor of the disc. In secondary and consecutive atrophy the pallor of the disc as a rule is much greater than in the primary form, in which latter condition the prevailing tint of the disc is grey or greyish-white, with well-marked circumference and normal-sized vessels.

The atrophy when advanced may cause total blindness, as seen sometimes in tabes; more usually there is but partial loss of sight, as seen in diminution of the sharpness of vision, with contraction or alteration in the field and loss of colour-perception.

DISEASE OF THE CHIASMA AND OPTIC TRACT

If the optic nerve be traced backwards from its origin in the ganglion-cells of the retina, its fibres are found to undergo partial decussation in the chiasma. The median fibres cross to the opposite side, forming part of the optic tract of that side; whilst the outer or lateral fibres pass directly backwards without crossing, and form a part of the optic tract on the same side of the median line. The left optic tract consequently contains fibres arising from the outer or temporal side of the left retina and fibres from the inner or nasal side of the right retina.

If the tract be followed backwards, it will be found to terminate in the pulvinar of the optic thalamus, the anterior corpus quadrigeminum, and in the *lateral geniculate body*. Since most of the fibres terminate in the end-brushes of this latter body it is recognised as the primary visual centre. From its ganglion-cells fibres arise which pass through the posterior tip of the internal capsule, and, spreading out in the optic radiations of Gratiolet, end in the occipital lobe, in which region of the brain the centre for vision has been located in the neighbourhood of the cuneus and calcarine fissure. Keeping in mind the course of the visual path as thus roughly sketched, it is easy to see how injury or disease at any part may cause visual symptoms.

Hemianopia, hemiopia, or hemianopsia (for the terms are synonymous) means loss of vision or blindness of one half of the field of vision in both eyes from causes independent of disease of the retina. Thus disease of one optic tract will cause loss of vision in the opposite half of each field, the nasal half of one field and the temporal portion of the other being involved. This condition constitutes what is known as *homonymous* or *lateral hemianopia*. When the nasal half of each field or the temporal half of each field is involved, the condition is recognised as *heteronymous hemianopia*.

Attitudinal hemianopia superior or *inferior* is the term employed to denote the rare condition in which the inferior or superior halves of the field of vision are involved.

A source of confusion must be remembered and guarded against. The rays of light entering the eye cross as they pass through its media, so that blindness observed in the nasal half of the field of vision is really due to loss of sight in the temporal half of the retina.

Right lateral hemianopia implies loss of sight in the right half of the field of vision in both eyes—*i.e.* of the temporal field in the right eye and the nasal in the left.

Left lateral hemianopia implies loss of sight in the left fields—*i.e.* of the temporal in the left eye and of the nasal in the right. Thus these terms are synonymous with *homonymous hemianopia*. Temporal hemianopia implies loss of vision in the temporal or outer fields in both eyes, as already explained, by the crossing of the light-rays in the eye-media; it is the result of loss of power in the nasal side of each retina. In this condition it will be observed that the left half of one field (the temporal) and the right half of the other field (also the temporal) are involved; hence the condition is synonymous with *heteronymous hemianopia*, and the same remarks are true of the nasal variety.

In all cases the entire half-field may be blind, or only a portion of it may be involved.

Lesions of the chiasma cause *temporal hemianopia*, the outer portion of each field being involved in both eyes owing to the involvement of the *nasal* fibres from each retina, as these nasal or internal fibres are the decussating ones. The outer or direct fibres generally escape, unless in the very rare condition in which a lesion on each side of the chiasma would catch the direct fibres and permit the decussating strands to escape. In this latter case a *nasal hemianopia* might be produced by the destruction of the fibres coming direct from the temporal side of the retina.

The nature of the lesions met with in the chiasma varies—viz. a tubercular or syphilitic growth, interstitial inflammation, or the pressure of a distended third ventricle, as in internal hydrocephalus or the atrophy with secondary changes seen in

tabes, may each cause signs. The results vary with the progressive nature of the lesion, and if the entire chiasma becomes involved total blindness follows in both eyes. If the lesion involve the central part of the chiasma and extend outwards so as to involve the direct fibres on only one side, temporal hemianopia may remain in one eye, whilst the sight in the opposite globe is entirely lost. An extension of the lesion forwards to one optic nerve or backwards along the tract will cause similar results.

Lesions of the Optic Tract and Centres.—If on one side, as already stated, this will cause homonymous hemianopia; thus a small tumour pressing upon the right optic tract causes loss of function in the right half of each retina, producing blindness of the left half of the field of vision in each eye—*i.e.* of the temporal field in the left eye and of the nasal field in the right eye, owing to the crossing of the light-fibres in the eye-media, as above explained. A lesion anywhere behind the chiasma and between this and the cortex of the occipital lobe will produce the same effect. Thus it follows injury to the pulvinar in the optic thalamus, the anterior corpus quadrigeminum, the lateral geniculate body, the posterior extremity of the internal capsule, the radiating fibres, or the cortex of the occipital lobe at the calcarine fissure. The defective halves of the visual fields are obviously on the opposite side to that of the lesion. *Wernicke's sign*—hemipic pupillary inaction—if present, will show that the lesion is located in the tract between the chiasma and the anterior quadrigeminal body. This is elicited by causing a ray of light to fall upon the blind part of the retina; if the pupil does not contract the lesion is in front of the corpus. Should the pupil react, the lesion is behind this—*i.e.* either in the internal capsule or the occipital lobe.

The hemianopia is sometimes partial, only a portion of the half-field of vision being entirely blind; and this is more common when the lesion is situated in the occipital lobe. Variations, however, are marked in the amount of fibres which decussate in the chiasma in different individuals, and these may lead to erroneous conclusions.

The nature of the lesions which cause lateral hemianopia—*viz.* syphilitic and tubercular meningitis and tumour—is liable also to cause the implication of neighbouring parts, so that eye-palsies, aphasia, hemiplegia, and hemianæsthesia often are present along with it. The hemiplegia is always on the same side as the loss of vision; the patient turns his head slightly towards the dark part of the field in order that light may fall upon the unaffected part of the retina.

Hemianopia may be present in a transient form in migraine and cerebral hæmorrhage, but exceedingly rarely in hysteria. Colour-hemianopia (*hemiachromatopia*) sometimes occurs even

when there is no blindness to white light ; as a rule, the loss for colours corresponds to the blindness for white. The lesion which produces the hemianopia may also cause optic neuritis. The fields clear up from the centre, the peripheral loss remaining longest ; some loss of acuity in the central vision is common. The condition may last permanently, and when the lesion affects both occipital lobes total blindness results in both eyes.

In cortical disease of the posterior inferior extremity of the parietal and of the marginal and angular convolutions there is dimness of sight in the opposite eye from concentric reduction of the field of vision for white and colours. This constitutes *crossed amblyopia*, but the eye on the same side is always also slightly involved. It may be produced by organic causes or may be associated with the hemianæsthesia of hysteria. Organic lesions causing this condition are often associated with *mind-blindness*.

Amblyopia is the term used to designate dimness of vision ; it is regarded as functional in character by most authorities, since there are usually no ophthalmoscopic changes, and the condition is often transient. To this category, as already stated under 'Retrobulbar Neuritis,' belong the toxic amblyopias (p. 1142). When the blindness is complete and bilateral, as in uræmia, in which it may come on with marked suddenness, the term *Amaurosis* is used.

Reflex amblyopia is the term employed to describe the dimness of vision arising from irritation of the fifth nerve in neuralgia or in dental caries. In hysteria the typical visual disorder is contraction of the visual fields and alterations in the perception of colour ; but a complete loss of sight in one eye has been occasionally observed. In all the cases of toxic amblyopia it must at present remain undecided whether the origin is a retrobulbar neuritis or an inhibition of the cortical visual cells.

LESIONS OF THE RETINA

Only the condition which is dependent upon general blood-diseases falls under the province of the physician. Inflammation of the retina, or retinitis, occurs so often in Bright's disease that it is usually designated *albuminuric retinitis*. The obvious changes, as demonstrated by the ophthalmoscope, consist of numerous small hæmorrhages in the nerve-fibre stratum of the retina ; these may exist alone or be accompanied by much swelling ; sometimes they are associated with papillitis (neuro-retinitis). The hæmorrhages, when recent, are bright red and often flame-shaped ; they are accompanied by spots of shining whiteness, especially numerous in the region of the disc. These silvery spots are highly characteristic ; they are the result of degenerative changes in the

retinal nerve-fibres and corpuscles. The hæmorrhages may almost entirely disappear, but usually more or less loss of vision remains from opacities and subsequent atrophic changes in the disc and macula lutea.

Albuminuric retinitis is commonly associated with arterio-sclerosis, and the tendency of the latter towards cerebral hæmorrhage and thrombotic softening is marked, so that with an inflamed disc, retinitis, hæmorrhage, and white spots it may be for a time impossible to decide whether the ophthalmoscopic changes are due to intracranial gross lesions or retinal mischief. Similar retinal inflammations, with extensive hæmorrhages, may occur in various forms of anæmia, gout, valvular disease, leukæmia, malaria, lead-poisoning, and purpura. In septic conditions, as pyæmia and ulcerative endocarditis, the hæmorrhages may be observed to form around minute white or yellowish-white septic emboli in the capillaries of the retina.

Syphilitic retinitis is usually associated with choroid inflammation, constituting the more or less characteristic choroido-retinitis. In congenital cases the condition known as retinitis pigmentosa is occasionally observed.

Tubercular deposits, when seen on ophthalmoscopic examination, are situated in the choroid.

Pulsation in the retinal arteries has been frequently observed in aortic regurgitation and exophthalmic goitre.

Embolism of the central artery is sometimes seen as an isolated condition or in association with cerebral embolism. It is often the result of valvular disease, sclerotic changes in the vessels, and ulcerative endocarditis, and may cause sudden blindness. Aneurismal dilatations may sometimes be observed upon the retinal vessels in general arterio-sclerosis.

Functional affections of the retina rarely, if ever, occur; the toxic and hysterical amblyopia supposed to be of retinal origin are, as already explained, due to changes behind the nerve. Hypersensitiveness of the retina may occur in hysteria, but even then it is difficult to say whether the disturbance should be regarded as peripheral or centric. The condition in which objects are only distinctly seen when viewed in a dull twilight (*hemeralopia*), and the more common state in which a strong light, natural or artificial, is essential for clear vision (*nyctalopia* or *night-blindness*), are probably of toxic origin. Thus both conditions have been observed in scurvy.

OSTEITIS DEFORMANS

This is the title given to a rare disease first described by Sir James Paget—hence sometimes called Paget's Disease—which is characterised by peculiar changes in many of the bones, leading to remarkable deformities in the cranium, clavicles, thorax, and extremities.

Etiology and Pathology.—The disease first shows itself in persons past middle-life, males being more frequently affected than females. Its causation is unknown and its pathology doubtful.

The pathological changes seen in the bones are suggestive of an inflammatory action originating in the periosteum which causes great thickening and increase in their diameters, whilst at the same time the osseous tissue in the interior becomes rarefied by absorption in some places, whilst at others the bony canals are inlaid with new osseous deposits, and new concentric and circumferential lamellæ may be laid down. These inflammatory changes are accompanied by softening, which causes bending, leading to the characteristic deformities.

Signs and Symptoms.—The thickened clavicles become much curved and prominent; the softened ribs yield and produce a marked widening of the lower zone of the thorax, causing prominence of the lozenge or diamond shaped abdomen. The femora curve outwards and the tibiæ forwards, causing a marked bandy-legged condition; the curving of the spine, with the bowing of the legs, may reduce the stature very considerably. The thickening of the cranial bones is usually marked, and as the facial bones do not participate the face appears much smaller, and assumes the appearance of a triangle, with the chin as the apex. The head, being thus increased in weight, falls forward so that the chin may rest upon the sternum.

The general health is in no way affected save by the mechanical difficulty which may arise from the spinal or thoracic deformity, and the course of the disease is steadily progressive. The malady is uninfluenced by any method of treatment. Death may result from respiratory embarrassment, and occasionally from the development of malignant tumours which have been observed to supervene in some cases.

The signs can hardly be mistaken for those produced by any other disease; fractures do not occur, which differentiates the condition from *mollities ossium*, and the enlargement of *acromegaly* is always confined to the bones of the face, hands, and feet.

OSTEO-ARTHRITIS.

(See ARTHRITIS DEFORMANS, p. 69.)

**OSTEO-ARTHROPATHY, HYPERTROPHIC
PULMONARY**

This rare affection is characterised by a marked symmetrical enlargement or hypertrophy of the hands and feet and of the carpal ends of the radius and ulna, as well as of the corresponding portions of the tibia and fibula, which enlargements are almost always associated with advanced disease of the lung or pleura.

Etiology and Pathology.—Empyema, fibroid phthisis, congenital heart-disease, malignant disease of the lung, and chronic bronchiectasis stand in some mysterious etiological relationship with the disease, which is therefore called hypertrophic pulmonary osteo-arthropathy. Of the cases hitherto recorded the majority (86 per cent.) were in males, mostly of middle or adult age; in a few of these there was a syphilitic history.

The pathology is obscure. Marie, who gave the above cumbersome name to the disease, regarded it as the result of a toxæmia from the lung, and it has been supposed to be of tubercular origin. It is sometimes described under the titles Marie's Disease, Secondary Hyperplastic Osteitis and Tubercular Polyarthrititis. Its proper place belongs to the category of the osteopathics referred to in the succeeding article, and Symes Thompson recognises two distinct forms or clinical types of the disease—the osteopathic and the arthropathic—in his recent analysis of eighty-eight reported cases. This authority regards the essential factor as a toxic focus somewhere in the body, with a condition in the lungs which interferes with the normal respiratory interchange.

Pathological changes have been observed not differing much from those seen in the bones and joints in osteitis deformans and in tabes. The knee and elbow joints have been found the seat of synovitis, with eroded cartilages. The soft tissues over the enlarged bones are not affected.

The condition is a very chronic one; occasionally it has been noticed to recede or disappear, but as most of the diseases with which it is associated are of a fatal nature it only possesses a pathological interest.

Symptoms.—The enlargement is characteristic inasmuch as it affects the *length* and thickness of the fingers and nails, especially deforming the terminal phalanges, whose extremities are much clubbed, whilst the carpus and tarsus are but

moderately increased in size, though the wrists and ankles are considerably swollen. In all these respects it is in marked contrast with *acromegaly*, and differs also from this affection in not involving the bones of the face and in never being associated with mental, speech, or ocular disturbances, and in the absence of the marked thickening of the skin. Occasionally there is some enlargement of the ends of other long bones, and sometimes kyphosis.

OSTEOMALACIA. (See MOLLITIES OSSIUM, p. 1045.)

OSTEOPATHIES

The trophic or trophoneurotic lesions of bone occurring in tabes, syringomyelia, and sometimes in general paralysis and leprosy, have been grouped together under the above title. They have been referred to under 'Ataxia.' The clinical signs are those of a *spontaneous* and *painless* fracture, which unites under mechanical treatment like an ordinary fracture, save that usually a much larger amount of callus is thrown out in the neighbourhood of the breach of continuity. The brittleness is seldom confined to a single bone, so that when this form of neurotrophic affection manifests itself the fractures are usually multiple.

The condition is so frequently associated with joint-lesions that the term 'Osteo-arthropathies' practically includes the group, as the majority of such spontaneous painless fractures occur in the limbs of tabetic patients in the neighbourhood of joints which are the seat of peculiar and characteristic changes, the term 'arthropathies' being employed to designate the neuropathic lesions affecting the joints in tabes and other affections.

The Arthropathies constitute a group with the same trophic or neuropathic origin as the Osteopathies; they frequently manifest themselves, as already mentioned in the description of locomotor ataxia, without any *symptoms* save the anæsthesia of the joint. The *signs* of the tabetic joint-lesion need not be again detailed; the sudden onset, abrupt swelling, and rapid destruction of the articulation, with or without hypertrophic changes, are the striking features, and it is noteworthy that the great majority of them occur in the lower extremity. Upon the whole, fracture occurs seldom in the tabetic trophoneuroses, though in all cases the atrophic changes are not confined to the articulation, but are found in the bones, chiefly of the type of an eccentric atrophy or a rarefying osteitis. When the vertebral column is involved the lesion may be either osseous or arthritic or both, so that Turney

classes the neuropathic affection of the spinal column as an osteo-arthropathy.

The pathology of the osteopathies, arthropathies, and osteo-arthropathies is identical; they are the result of a trophoneurosis. The afferent or sensory fibres which supply the joint probably convey stimuli to the posterior nerve-root, and by reflex action these stimuli are conveyed to the vaso-motor centres regulating the nutrition of the articulation. These sensory fibres, according to the above theory of Marinesco, as stated by Turney, 'form the afferent path of a reflex arc, the efferent portion of which is provided with nerves having a vaso-motor function.' That the changes are the result of some disturbance in a supposed medullary centre he considers disproved by the fact that the arthropathies always occur in the joints supplied from the diseased cord at certain levels. The subject is one of great interest and practical importance, since arthropathic changes are not confined to tabes, syringomyelia, and general paralysis, but have been observed to follow injury and disease of nerves, cord, and brain; and they are not infrequently met with in various forms of insanity and also in syphilis, as recently described by Morestin. It can scarcely be doubted that many obscure joint-affections usually regarded as of rheumatic origin are examples of true neuropathic origin.

It should be remembered that the hypertrophic pulmonary osteo-arthropathy described upon page 1149 really belongs to this group of trophoneuroses, one type of case, in which the bones are chiefly involved, falling under the osteopathies, and the other, in which the joint-affection predominates, belonging to the arthropathies, though apparently the great majority of the hitherto recorded cases might be classed under the mixed form of osteo-arthropathies.

OXALURIA

This is the name given to the condition in which oxalates are found in excess in the urine. Not more than one-sixth to one-third of a grain of oxalic acid is excreted daily by the normal individual. When this substance is found in oxaluria, or the so-called oxalic-acid diathesis, it appears in the form of dumb-bell or octahedral crystals of oxalate of lime; in normal urine it is held in solution by the acid sodium phosphate. Oxaluria of a temporary or transient character is due to the ingestion of certain vegetable substances. Thus rhubarb-stems when freely indulged in, as in the form of stewed rhubarb or tart, often cause a serious irritation of the bladder, pelvis of the kidney, and urethra, owing to the mechanical injury of the mucous membrane by the angular gritty crystals of

oxalate on their elimination. Onions, cabbage, and tomatoes cause less intense deposits, which may not appear till the urine has cooled for some hours.

The term 'oxaluria' should be restricted to those cases where the salt is persistently excreted. Its origin in such cases is doubtful, but it is held by many to be due to retarded oxidation of carbohydrates, and to occur in those conditions in which the formation of free hydrochloric acid is diminished and where the intestinal digestion is retarded. In health the traces of oxalates usually present are supposed to come from the food.

Its frequent association with the gastric and other neuroses, neurasthenia, spermatorrhœa, and hypochondriasis is indicative of some alteration in the normal metabolic processes. Though it is by no means certain whether the oxaluria is the cause or the result of the neurotic disturbance, nevertheless the symptom-complex is by many regarded in the light of a special diathesis, as the presence of uric acid has led to the term 'uric-acid diathesis' as a synonym for gout.

It must, however, be remembered that excessive formation and deposition of oxalates may long persist in a latent form, giving rise to no symptoms till the formation of a mulberry calculus causes its own mechanical disturbances. A considerable degree of oxaluria may be associated with lithæmia, gout, or albuminuria. It has been observed to occur in cases of extensive burns, and is not rare in conditions caused by organic brain-lesions and in severe examples of mental overstrain and insomnia.

The treatment of oxaluria in the transient cases caused by dietetic errors is obvious, as the condition disappears rapidly upon the removal of the cause. Where it is associated with the symptoms of a neurosis, the general treatment indicated in the management of neurotic conditions should be pursued. This consists in the setting right of any known violation of health laws, the regulation of hours of rest and exercise, and a revision of the dietary of the patient. Of most importance is active open-air exercises; and where these cannot be indulged in for any special reason, massage and gymnastics should be recommended.

In the neurasthenic cases moral treatment is indicated; and where the stomach or dyspeptic disturbances are prominent, digestives combined with free hydrochloric acid are useful; or should organic acidity be troublesome, as it often is, alkalies are indicated.

OXYURIS VERMICULARIS

This common intestinal nematode is known as the thread-worm. The female is found in the fæces as a small wriggling body resembling a piece of white thread, in length not more than one-third, or at the most half an inch, with a filiform tail. The male worm is seldom seen, as it perishes after the completion of the sexual act ; it is about one-third the length of the female, and has a truncated or curled tail. The female, when fully matured, is found filled with ova ; these are minute oval bodies flattened on one side.

The ova find their way into the human stomach in drinking-water, or on raw fruit or salads ; the embryo is liberated by the action of the gastric juice, and on becoming impregnated in the small intestine descends to the large bowel, and ultimately passes out in the fæces. The worm whilst in the lower part of the rectum causes intense itching by its migration, especially at night, when it is liable to wriggle out through the anus. Finding its way into the vagina, it may cause severe irritation in this region also ; bladder-irritation, emissions, and various reflex nerve-disturbances are not uncommon. It has even been known to penetrate the intestinal wall and cause localised suppurative peritonitis or abscess, though this result is very rare. The pruritus caused by the parasite may induce most severe insomnia, and the scratching is liable to excite troublesome eczema. The ova, getting under the free margin of the finger-nails, are liable to find their way back again into the mouth and stomach, especially as the parasite is most common in children. This is the explanation of those cases where constant recurrences occur after treatment, the patient from time to time re-infecting himself during sleep.

The treatment is simple and efficacious if due care be taken to avoid self-infection by such means as shall prevent the child from soiling its fingers and conveying them afterwards to the mouth.

As the habitat of the worm is in the lower part of the bowel, it may be easily reached by enemata, the most simple being one of chloride of sodium in water. These should be continued every few nights for a month, in severe cases, as long as the parasites or their ova continue to appear. Infusion of quassia is highly efficacious, and the salt may with advantage be dissolved in it. Santonin internally has been used, but owing to the liability of its being entirely absorbed before reaching the cæcum it is not so certain as naphthalin. A little conium or mercurial ointment applied around the anus will prevent the night-migrations of the worm, and diminish or prevent itching.

PANCREAS, DISEASES OF

As the result of pathological observation, much has of late years been made known of the nature of structural affections of this gland, but upon the whole little has been established clinically. Often extensive disease is accidentally discovered after death where no symptoms during life pointed to the pancreas as the seat of any diseased action, and the writer has seen unmistakable evidence during life of the whole of the pancreatic juice being prevented entering the duodenum owing to rupture of the main duct, without any symptom being produced beyond the mechanical accumulation of enormous amounts of the secretion in a cyst-like cavity. Removal of the entire gland might possibly be unattended by symptoms of any kind, unless neighbouring organs were injured in the operation, since all the functions of the pancreas, as pointed out by Mayo Robson, with the doubtful exception of the glycogenic, may be vicariously discharged by other organs.

HÆMORRHAGE INTO THE PANCREAS

Etiology and Pathology.—Apart from any history of trauma or the existence of abnormal blood-conditions like those seen in purpura, hæmorrhage has undoubtedly occurred even in apparently healthy subjects, and has been in some cases the cause of death. The condition has been supposed in most instances to be the *result* of an acute inflammation of the whole or of a part of the gland—hence the introduction of the term *hæmorrhagic pancreatitis*; by some the hæmorrhages have been regarded as the *cause* of the inflammation.

As might be anticipated, pancreatic hæmorrhage is more likely to be associated with those diseased conditions causing blood-stasis, as cirrhosis of the liver, valvular disease, arterial sclerosis, chronic pulmonary affections, embolism of the pancreatic vessels, and rapidly growing cancers. It is also liable to follow in severe altered conditions of the blood, as in septic states, purpura, scurvy, &c.

Fat-necrosis in the pancreas or in its immediate vicinity has been often found associated with hæmorrhages, whether as the cause or result of these is still by no means clear. Trauma is an obvious etiological factor.

Symptoms.—The disturbances caused by pancreatic hæmorrhage depend largely, if not entirely, upon the amount and rapidity of the flow of the extravasated blood. When the hæmorrhage is gradual no symptoms or signs may be visible, as has been proved in cases where the remains of even considerable extravasations have been found in the pancreas of patients dying from some other affection. In sudden

hæmorrhage, severe pain above the umbilicus, with vomiting, restlessness, collapse, and speedy death, will supervene as in any other form of internal abdominal bleeding, and the extravasated blood may be found to have forced its way into the loose tissue behind the peritoneum, or even filled its small sac.

ACUTE INFLAMMATION OF THE PANCREAS

Etiology and Pathology.—This condition may be caused by any of the previously mentioned etiological factors which sometimes lead to hæmorrhage. Trauma, obstruction of the common duct by gall-stones, catarrhal swelling, cancer, pancreatic calculi, or the entrance of round worms, or the occlusion of the duct by inflammatory adhesions caused by duodenal ulcers, &c., may all produce pancreatitis, and, as already stated, the inflammation may be the result of a simple hæmorrhage produced by some unknown cause. Acute pancreatitis has been observed to occur in mumps; in one case seen by the writer an acute and almost fatal inflammation of the pancreas followed a mild attack of parotitis in a middle-aged woman. Simple or malignant ulceration of the posterior wall of the stomach may also invade the pancreas and excite acute inflammation of its parenchyma. The type of the inflammation may be *catarrhal* when the mischief arises in the duct or in the duodenum or stomach, as in cases of catarrhal jaundice. The introduction of pus-forming germs is liable to alter the type of a simple catarrhal pancreatitis into a *suppurative* variety.

Of *parenchymatous* inflammations of the pancreas, Mayo Robson recognises the *diffuse suppurative* and the *gangrenous* forms resembling those occurring in acute appendicitis, the *ultra-acute hæmorrhagic* form in which the hæmorrhage precedes the inflammation, and the *acute hæmorrhagic* type in which less extensive hæmorrhages are preceded by inflammatory action.

Symptoms.—The *catarrhal form* is usually associated with the symptoms and signs of the primary disorder which causes the inflammatory swelling of the duct. As this is usually the result of some mischief in the region of the ampulla of Vater, icterus is present, as is also gastric derangement with marked loss of appetite, nausea or vomiting, constituting the usual clinical picture of ordinary catarrhal jaundice.

When catarrhal pancreatitis is the result of cholelithiasis, there are added the symptoms and signs met with in gall-stone attacks, in which the impaction occurs at the lower extremity of the common duct. In these cases, bile finding its way along the partly occluded duct may enter the canal of Wirsung and excite a severe attack of hæmorrhagic pancreatitis, as proved by the experiments of Opie. Symington has described considerable variations in the duct, and in one case

the upper part of the duodenum was encircled by a ring of pancreatic tissue; the accessory duct of Santorini usually opens directly into the duodenum above the common orifice of the bile and pancreatic main duct. These variations in the termination of the duct may exercise a serious influence in diseased conditions, involving their orifices, especially as regards the following:

Suppurative catarrhal pancreatitis is usually associated with the presence of gall-stones in the common duct, or it may be the result of duodenal catarrh in which pus-forming organisms enter the accessory or main duct of the pancreas. The symptoms will resemble those of suppurative cholangitis and those caused by pus in the gall-bladder; the condition belongs to the province of the surgeon, and is only amenable to operative treatment, which, at the same time, provides for drainage of the bile-ducts. This latter procedure is insisted upon by Mayo Robson, even when in catarrhal pancreatitis no gall-stones or pancreatic calculi are found.

Though the simple catarrhal type of inflammation usually resolves, if there be evidence of continued failure in weight, and the other signs of the supervention of interstitial inflammation about to be mentioned, the case passes into the domain of operative surgery.

Interstitial Pancreatitis of the acute types, as already stated, is generally of the hæmorrhagic variety. It is sometimes associated with alcoholism, and often with cholelithiasis. It occurs most frequently in males before or about middle-life; occasionally it is observed to occur in women at the lying-in period. The pathological changes in the gland are those met with in other acute parenchymatous inflammations, save that the tendency to hæmorrhages into the gland-tissue is usually much more marked and extensive, and evidences of fat-necrosis in the gland and peritoneum are often present.

Symptoms.—In the *ultra-acute variety* the symptoms are those of an abrupt and extensive internal hæmorrhage ending in collapse and a speedy fatal termination.

In the *ordinary acute variety*, where the inflammation has preceded the hæmorrhages into the glandular parenchyma, the attack also commences suddenly, but with less abruptness and severity. There is severe abdominal pain in the epigastrium, soon followed by vomiting, and associated with thready pulse; swelling in the upper abdominal zone is observed with signs of a tumour of the pancreas, which is very painful and sensitive to pressure, and is accompanied by greatly increased muscular resistance; jaundice may occur. The collapse at first may be associated with a sub-normal temperature, but at a later stage slight fever sets in. The disease usually runs a rapid course, and may eventuate in a fatal issue.

within a few days. Usually there is not time for the establishment of the other signs which are often found in association with destructive disease of the pancreas, as the presence of fatty stools, glycosuria, emaciation, &c. Cammidge, by a modification of the phenyl-hydrazin reaction, obtains yellow crystals from the urine after hydrolysis by boiling with hydrochloric acid, which he considers as pathognomonic of disease of the pancreas; but his results have been recently disputed by Ham, Cleland, Gruner, and Willcox. Profuse salivation (sialorrhœa) sometimes occurs in acute pancreatitis.

Diagnosis.—Hæmorrhagic pancreatitis may be readily mistaken for *acute intestinal obstruction* or *perforative peritonitis*, vomiting, collapse, constipation, and abdominal distension being prominent symptoms in these conditions. The pain in intestinal obstruction is more decidedly colicky and rhythmical in its nature, being usually produced by the exalted vermicular contractions occurring above the seat of obstruction. The pain in acute inflammation of the pancreas, though also of intense severity, is more constant in character from the mechanical injury to the cœliac plexus caused by the extensive hæmorrhagic infiltrations. In perforative peritonitis the history of a gastric ulcer or of appendicular mischief is often obtainable.

Prognosis.—This is always grave, the disease in acute cases tending to end in fatal collapse; but laparotomy has proved successful in a limited number of cases, even when the inflammatory action had eventuated in abscess.

Gangrenous Pancreatitis is usually a sequel to the acute hæmorrhagic type.

Symptoms.—The initial symptoms and signs are those already described, but in the rare cases of acute pancreatitis which do not speedily end fatally the supervention of rigors and high irregular temperature may be accepted as indicative of abscess or necrosis of the organ. A portion of the pancreas, or the entire gland, may be converted into a dark, firm, necrotic mass surrounded by a wall of sloughing tissue and presenting the characteristic appearances seen in gangrenous appendicitis. The necrosed organ may occasionally be felt as a tumour above the umbilicus.

Prognosis.—This is always grave, but death does not occur till a much later period than in the acute hæmorrhagic type, probably because the condition is only likely to supervene in those cases in which extensive hæmorrhage does not take place, so that life may be prolonged beyond a fortnight after the onset.

Treatment.—Laparotomy, followed by the removal of the necrosed portion of the gland and the establishment of suitable drainage, has been successful, and a similar result

has been obtained in those cases where gangrene of the organ has been the result of a perforating ulcer of the stomach or duodenum.

CHRONIC PANCREATITIS

Pathology and Etiology.—This starts as an interstitial inflammation, whose proliferated cell-elements develop into fibrous tissue, which, in the *interlobular* form, ultimately causes pressure upon the glandular elements in the pancreas, and so interferes with its digestive function that fat appears in the stools and emaciation occurs. There is an *interacinar* variety of interstitial pancreatitis recognised by Opie, in which the fibrous tissue invades the islands of Langerhans, the result being that the metabolic function of the pancreas is diminished or abolished, and diabetes or glycosuria results, in addition to the disturbances of digestion.

The early stage of interstitial chronic pancreatitis may show a considerable hypertrophic change in the organ, but as the process advances the pancreas becomes firm, tough, and atrophied, like the liver in alcoholic cirrhosis; hence the condition is often described as pancreatic cirrhosis or sclerosis. The disease may be the result of almost any of the causes which produce acute hæmorrhagic pancreatitis; it is, however, much more frequently caused by the blocking of the common duct by gall-stones, or of the pancreatic ducts or canal of Wirsung by calculi, and the subsequent infective processes which are liable to follow. In these cases the affection, though regarded as primary, is an extension of the catarrhal inflammation from the ducts to the parenchyma of the gland. The whole or a part of the pancreas only may be involved. Mayo Robson recognises a secondary form due to syphilis, alcoholism, arterial sclerosis, or to the infective organisms of typhoid fever and influenza.

Symptoms and Diagnosis.—The symptoms are those of obstruction of the duct in the region of the ampulla of Vater, so that the pancreatic affection may be overshadowed by intense jaundice. A *malignant growth* in the head of the pancreas may effect complete obstruction, ending in deep jaundice and enlargement of the gall-bladder, a condition which is undistinguishable from chronic interstitial inflammation of the same portion of the gland, even after the abdomen has been opened and the diseased tissue is actually in the hand of the operating surgeon. A distended gall-bladder with secondary nodules in the liver will obviously demonstrate the condition to be a malignant one. The presence of fatty diarrhoea, of retardation of the digestion of albuminous foods, diabetes or glycosuria, and the discovery of leucin and tyrosin in the urine, taken together with a firm tumour in the region of the pancreas in a subject between thirty and forty, though they

do not exclude cancer, are upon the whole more likely to be caused by interstitial pancreatitis. Marked emaciation characterises both conditions. A *gall-stone* impacted in the ampulla of Vater usually cannot be differentiated; the pain and tenderness may, however, aid in the diagnosis, being in gall-stone cases localised in the region of the gall-bladder or radiating towards the right side of the thoracic margin or right shoulder, whilst in pancreatic disease pain is usually limited to the pancreatic region, and remains localised posteriorly about the spine. In pancreatic disease bile may be present in the motions, but the absence of the secretion of the pancreas often causes them to be almost clay-coloured, as well as greasy, the normal pigment being the result of a combined action of the bile and pancreatic secretion on the fæces, as maintained by Walker. Sialorrhœa is very occasionally observed.

Prognosis.—This is obviously as difficult and uncertain as the diagnosis, but the supervention of acute glycosuria may be accepted as evidence that a fatal issue is not far off. Some cases of chronic sclerosis or cirrhosis of the pancreas have been known to exist for years. The prognosis in many instances is distinctly favourable when the case falls under the category of the operative class.

Treatment.—Where interstitial pancreatitis is diagnosed, the strong probability of the disease being due to obstruction from gall-stone or pancreatic calculi should encourage the surgeon to insist upon early operative treatment as holding out the only hope of cure or amelioration. The removal of the obstruction from the pancreatic or common bile duct should be undertaken before the interstitial inflammation is permitted to invade the islands of Langerhans. The operation consists in the removal of the calculi and the thorough drainage of the bile-ducts by cholecystotomy or cholecyst-enterostomy. Moynihan insists upon the establishment of drainage, even when no obstruction has been discovered. The oozing of blood in jaundiced cases is a serious complication, which, however, may be successfully forestalled or met by large doses of calcium chloride. The administration of pancreatic extract in the form of a keratin-coated capsule may be employed in all cases with advantage before operation, and also in cases unsuitable for operation.

FAT-NECROSIS

The presence of this condition in the various forms of inflammation of the pancreas has been already incidentally mentioned. Opie has produced fat-necrosis in the organ by ligaturing the pancreatic ducts in the cat, and also by injecting bile into the canal of Wirsung in the dog. The fatty tissue in the pancreas and surrounding organs, including the sub-

peritoneal layer, the pleura, the heart, and even the subcutaneous tissues of the abdominal wall or more remote regions, is disintegrated or necrosed by the infiltration, by way of the lymphatic stream, of the fat-splitting ferment (steapsin) from the pancreas.

The fat is split into fatty acids and glycerin. As the latter is readily absorbed by the vessels, the acids crystallise or combine with the lime-salts of the blood, forming the small yellow areas first described by Balser and found in the pancreas, or in the omentum in the immediate vicinity of the pancreas. The presence of fat-necrosis in the abdomen is a valuable indication of pancreatic disease in the initial stage of laparotomy. It is very constant in acute hæmorrhagic inflammation of the organ, and when not very obvious it can be demonstrated by Bender's plan of applying a solution of acetate of copper to the tissues.

The prognosis in acute pancreatitis is rendered more serious by the evidence of extensive fat-necrosis, but many cases of pancreatitis in which this sign was noticed during the laparotomy have been known to recover.

TUMOURS OF THE PANCREAS

These, if we exclude cysts, are usually of the carcinomatous type, but, compared with malignant disease of the liver and stomach, primary cancer of the pancreas is rare. Sarcomata and other rarer types are very seldom seen. The scirrhus form of carcinoma is most frequently met with; usually it is situated in the head of the gland, and in thin subjects a hard nodular tumour, which does not move in respiration, may be palpated through the abdominal wall.

The signs and symptoms of pancreatic cancer are of great importance, because of the liability of the disease to cause jaundice, which must be differentiated from the other forms of obstructive jaundice. The chief points in the diagnosis are already discussed under 'Jaundice,' upon page 747, the most important being the presence of great distension of the gall-bladder, generally without ascites. The anatomical position of the diseased organ lying in front of the aorta must be remembered, as the conveyed pulsations may simulate abdominal aneurism. The prognosis is obviously a fatal one, though occasionally operative treatment has been reported as successful.

Cystic tumours are caused usually by obstruction of the ducts or of the canal of Wirsung by a calculus or cicatricial narrowing; such a *retention*-cyst may reach a considerable size, forming a spherical tumour projecting into the small peritoneal sac. It may push the stomach upwards, and finally come into contact with the abdominal wall above the colon or even below the umbilicus, or it may remain behind both of these

viscera, or in rare cases it may project immediately below the diaphragm, and, pushing the stomach downwards, remain as an obscurely felt enlargement high up in the abdominal cavity. Interstitial pancreatitis commonly, and malignant disease more rarely, by blocking the smaller ducts, may cause alveolar distension, ending in single or multiple retention-cysts. Occasionally a true *proliferation*-cyst may be caused by a neoplasm of the adenomatous type. Hydatid cysts are very rarely met with.

Pancreatic cysts may remain for many years without causing any symptoms save those arising from their pressure upon neighbouring organs. In those of rapid growth there may be considerable digestive disturbance or vomiting, associated with intense pain. Glycosuria, fatty diarrhoea, and greasy clay-coloured stools, with marked emaciation, only occur when the disintegration of the gland has become extensive; sialorrhoea is rare.

Diagnosis.—*Cystic disease of the kidney, hydronephrosis, and hydatid disease* of other organs are as a rule differentiated from pancreatic cysts by an examination of the fluid drawn off by a fine aspirating needle. The pancreatic cystic fluid varies considerably in colour; it is usually turbid and brownish; the writer has seen it almost as clear as water, and when blood is present it may be bright red, of the colour of coffee-grounds, or even black. The s.g. is usually between 1012 and 1020, the reaction is alkaline, albumin is always present and sometimes cholesterin, and generally the pancreatic ferments can be recognised easily by the usual physiological tests. The fluid will change starch into sugar, emulsify fat, and convert proteids into peptones, when all three ferments are present. More commonly, however, but one or two of these are found in the fluid, and, as pointed out by Osler, the digestion of albumin and fibrin is the only positive sign of the pancreatic origin of the liquid, owing to the fact that the emulsifying and diastatic ferments are frequently found in other exudations.

Ovarian cysts are usually excluded without difficulty, owing to the high and central position of the tumour in the abdomen.

Treatment.—Incision with the establishment of efficient drainage is the recognised procedure in all cases.

There still remain for notice the so-called *pseudo-cysts* of the pancreas, which are usually the direct result of injury causing the accumulation of fluid in some portion of the loose tissue of the pancreas, or of a neighbouring organ, or in the small sac of the peritoneum. In one typical case, published elsewhere by the writer, a large tumour, closely simulating an aneurism, formed after a severe localised injury to the abdomen; he tapped the tumour, the contents of which were found by Professor Matthew Hay to consist of pure pancreatic juice. Gallons of this fluid were removed

from time to time, but the patient did not suffer from any of the symptoms supposed always to follow the arrest of the secretion of the gland, though the enormous quantities of fluid, possessing in a very active form all the physiological qualities of undiluted pancreatic juice, continued to be removed by the aspirator for many weeks. The tumour, after one of the tappings, rapidly filled up with a bloody liquid. Symptoms of peritonitis supervened, but the patient made a rapid and complete recovery and remained perfectly well.

PANCREATIC STONES

These calculi are so rare that they may be regarded as mainly of pathological interest. Mayo Robson believes that they never form in a healthy pancreas, but are the result of a catarrhal inflammation of the ducts, causing stagnation of the secretion of the organ. They are composed of lime-salts, chiefly carbonate, with some phosphates, and are consequently opaque to the X-rays, which pass through the calculi of biliary origin. They may be very numerous; very rarely does only one exist. By their obstruction of the ducts, retention-cysts and interstitial pancreatitis may be produced, by which results their presence may be suspected. A few cases have been reported where paroxysmal pain like that observed in gall-stone colic has occurred, followed by the passage of numerous small calculi and some sand-like concretions in the stools. In operations for the removal of a degenerated or cirrhotic pancreas these calculi are occasionally met with, and it is probable that they are the result of the primary disease, and not the cause of it, as usually supposed. When symptoms direct attention to the pancreas, and the X-rays show calculi to be present, removal by operation may be indicated.

PARALYSIS

This term is usually confined to loss of power in the voluntary muscles, but, since the loss of power in transmitting sensory impressions is sometimes called *sensory* paralysis, the term *motor* paralysis is used in contradistinction.

The loss of power may be complete or incomplete, all degrees being met with in diseases of the nervous system, the minor or incomplete degree of weakness or loss in voluntary movements being often styled *paresis*. In the great majority of all forms of paralysis the cause is due to lesions in the nervous system. In pseudo-hypertrophic paralysis, and in some forms of muscular atrophy, the lesion may be due to degenerative changes in the wasted muscles, the conducting path in the cerebrum, spine, and motor nerve-trunks being

intact. In a similar manner the loss of muscular power may be due to defective nutrition of the muscles, the result of inanition; but this form of muscular weakness is not usually included under the term 'paralysis.'

The lesion causing the motor palsy is very often the result of a gross organic disease or injury which prevents the transmission of impulses from the cerebral cortex; but the remarkable paralytic phenomena occurring in hysteria are notable exceptions. In this condition the paralysis is variously regarded as due to inhibition, to loss of the proper balance normally present in the higher cerebral functions, or to some *molecular* alteration in the cells of the grey matter in the cerebral cortex. Owing, however, to the absence of all anatomical proof of organic changes, hysterical paralysis must still be regarded as of the type of a purely functional affection.

The various forms and varieties of paralysis are usually classified under either one or other of three distinct types—*i.e.* the *cerebral*, *spinal*, or *peripheral*; but, as will be described under 'Paralysis, Spastic,' strictly speaking only the cerebral and spinal types should be recognised.

In *cerebral paralysis* the lesion may be of the nature of a laceration or of a mechanical compression of the nerve-fibres, of a shutting off of the blood-supply or of an inflammation or degeneration involving the cerebral tissue. Of whatever nature, the lesion effects paralysis by its injury to some part of the motor path in the brain, either at its origin in the cortex or as it proceeds downwards through the centrum ovale, internal capsule, crista of the cerebral peduncle, and pons to the anterior pyramid in the medulla.

Cortical lesions usually produce a limited paralysis, or more commonly a paresis confined to one section of the body on the opposite side to the lesion. Such paralysis is usually called a *Monoplegia*; thus a lesion in the cortex of the anterior central convolution at its lower part will cause paralysis of the face and tongue, and if the arm-centre be also involved the upper limb is weakened or some of its muscles only affected. The facial centre may alone be involved, causing a facial monoplegia, or only the centre for the thumb being affected the loss of power is confined to its muscles. The involvement of the leg-centre in the ascending frontal and parietal convolutions in the highest part of the motor area will produce a *crural monoplegia*.

The most common form of cerebral paralysis is that due to injury or disease affecting the motor path lower down, especially in the region of the posterior limb of the internal capsule. This causes *hemiplegia*, as described under 'Cerebral Hæmorrhage' (p. 177), in which the lower part of the face, the arm, tongue, and leg, are paralysed upon the opposite side of the body.

The chief characteristics of cerebral paralysis are thus seen in a motor disturbance, usually confined to one side of the body, the trunk-muscles escaping or being but slightly weakened, and the sphincters remaining intact. The cutaneous sensibility is usually lessened in the earlier stages over the affected half of the body; the electrical reactions of the paralysed muscles are practically unaffected; the deep reflexes are increased and the superficial ones diminished on the affected side, and often mental symptoms are present at the commencement or at some later stage of the attack. It is rare for a cerebral lesion to produce paralysis of both sides of the body, but bilateral lesions are witnessed in the condition known as pseudo-bulbar paralysis. True bulbar paralysis should be regarded as of the spinal type, being caused by disease of the lower neurons, though the lesion is in the medulla.

Spinal paralysis usually involves both sides of the body. The onset is not associated with mental symptoms or convulsions, and in the majority of cases the lower extremities are alone involved, constituting the usual form of *paraplegia*. The trunk-muscles suffer according to the level of the cord affected, and the upper limit of the motor paralysis is often marked by a painful girdle-sensation in those cases where the sensation is markedly affected. A characteristic feature is the liability to the impairment of the sphincters, as detailed under 'Myelitis (p. 1079). The deep reflexes are lost in complete transverse lesions, but in the usual type of the incomplete variety occurring in the dorsal region the reflexes are greatly exaggerated as secondary degeneration occurs.

The electrical reactions of the muscles vary in a similar manner; when the dorsal cord is affected they are normal, whilst in lumbar disease of the cord the muscles rapidly waste and show the reaction of degeneration. The arms are paralysed in addition to the legs when the cervical region is involved, and their reflexes may be absent whilst those in the legs are increased.

In rare cases the lesion causing spinal paralysis may be confined to one-half of the cord, in which instance the paralysis only affects one side of the body—that corresponding to the lesion—constituting the condition known as *Hemiparaplegia*, in which there are no mental symptoms or evidence of any weakness affecting the facial nerve—characteristics which distinguish it from hemiplegia, even when the unilateral spinal lesion involves one arm and one leg.

Peripheral Paralysis.—When pressure or injury to a nerve-trunk interferes with its function, the paralysis is at once recognised as of peripheral origin by the loss of motor power being confined to those muscles deriving their supply from the affected nerve. Rapid degenerative changes are set up in both the nerve and muscle, and the characteristic reaction of

degeneration is obtained. In the nerve the loss of electrical irritability to both the faradic and continuous current usually takes place rapidly. The muscles cease to respond to faradism, but their irritability to the continuous current increases; at a much later stage this increased irritability may be lost. This reaction of degeneration is usually complete in seven to ten days. The muscles rapidly waste.

It must, however, always be borne in mind that the reaction of degeneration is obtained not only in severe lesions of the peripheral nerves, as in traumatic cases and the toxic neuritis produced by lead, arsenic, alcohol, and the poison of diphtheria, but it is present when the anterior *horns* of the cord are diseased, as in infantile paralysis, amyotrophic lateral sclerosis, the spinal form of progressive muscular atrophy, and diffuse myelitis. The same result occurs in progressive bulbar paralysis where the nuclei of the nerves are diseased in the bulb, since these correspond physiologically and anatomically with the cells in the anterior horns of the cord. The reaction of degeneration is also obtained in disease or injury of the anterior *nerve-roots*, as in spinal caries and tumours, and in the compression caused by syphilitic growths and cervical hypertrophic pachymeningitis. The spinal and peripheral types of paralysis, whether caused by disease of the bulbar nuclei, cord disease, or affections of nerve-trunks, or their peripheries, should, strictly speaking, be all classed under the spinal type. (See 'Paralysis, Spastic'.)

The different forms of paralysis for convenience are described under the titles of the disease in which the loss of motor power is a symptom, as under 'Hemiplegia,' 'Neuritis,' 'Cerebral Hæmorrhage,' 'Cerebral Abscess,' 'Cerebral Tumour,' 'Hysteria,' 'Myelitis,' 'Meningitis,' &c. Only those forms of paralysis are here noticed which have not been detailed previously.

PARALYSIS, ACUTE ASCENDING

This affection, usually known as 'Landry's Paralysis,' is characterised by a flaccid paralysis commencing in the lower extremities and rapidly ascending till the trunk, diaphragm, arms, neck, and pharyngeal muscles are involved, often terminating fatally in a few days from paralysis of respiration.

Etiology.—Little is known definitely of the cause of the disease. It occurs most frequently in males between the ages of twenty and forty years. Syphilis, alcoholism, exposure to cold, and traumatism have been regarded as etiological features, but it is very doubtful if they should be considered as causative agents in any sense save that of their occasional action as predisposing factors. The disease has

been frequently observed to follow various infectious conditions, as typhoid fever, diphtheria, small-pox, influenza, pneumonia, and septic states, but in many instances no microbic or toxic agent can be traced.

Pathology.—The most careful investigation of the nervous system under the former methods of examination generally revealed no anatomical or pathological changes in the brain, spinal cord, nerves, or muscles of patients dying from this form of paralysis. Landry, who first described the disease, regarded the negative results of the *post-mortem* examination as characteristic of the affection. The newer histological and bacteriological methods have demonstrated in several cases the presence of various bacteria, as those of pneumonia and typhoid fever, staphylococci, and streptococci in the blood and central nervous system, but no *specific* organism has been up to the present isolated. Minute changes, chiefly of an inflammatory nature or produced by extravasations of blood, have been observed as disseminated foci in the grey matter of the spinal cord and medulla. Evidence of peripheral neuritis has been found by Goetz and Déjerine, Barth, Ross, and many other observers, but these afford no definite solution of the pathological problem.

The general opinion is that Landry's paralysis is of the nature of a toxæmia, probably caused by the introduction into the blood of some micro-organism whose chemical products act as a poison on the delicate fibres in the grey matter of the anterior horns, or on the motor tract in the medulla, or on the peripheral nerve-endings. The symptom-complex is best explained upon the theory of a multiple neuritis implicating at the same time the anterior horns, as seen in poliomyelitis. The theory of its being a functional affection or neurosis is no longer tenable.

Symptoms.—The paralysis begins in both legs, sometimes in one leg a few hours before the other. The loss of power may be complete in both legs inside twenty-four hours from the first moment of onset. The sensory disturbances are but slight, if present at all they are confined usually to a scarcely recognisable paræsthesia. The palsy is of the flaccid type without atrophy, the electrical reactions are normal, and the sphincters are intact, but the reflexes are speedily lost. The main characteristic of the paralysis is the rapidity with which it spreads upwards, involving the pelvic, abdominal, and thoracic muscles, and producing complete loss of power in both arms, sometimes within forty-eight hours, or even less, after the first signs of weakness in the legs have appeared. The neck-muscles succumb rapidly; the pharynx, palate, tongue, and lips become paralysed, as seen in difficulty of articulation and deglutition. Death usually results from asphyxia caused by paralysis of the diaphragm and thoracic

muscles, and the final issue may occur within two or three days; more frequently, however, it is postponed for a week from the onset. Fever is usually entirely absent throughout. Rare cases have been recorded, which in other respects conform to the Landry type, where the disease has appeared first as a bulbar paralysis, rapidly extending downwards to the arms and trunk. Enlargement of the spleen has been observed in several cases.

Diagnosis.—The greatest difficulty occurs in the elimination of *multiple neuritis* when this is of the rapidly progressive type, but the absence of anæsthesia and of symptoms first appearing in the hands and feet before the extension of the paralysis to the trunk usually suffice to exclude neuritis. There will be great difficulty in this differential diagnosis, however, in those very rare cases of Landry's paralysis where sensory symptoms, as tingling, numbness, or hyperæsthesia are prominently present at the onset.

Meningeal spinal hæmorrhage is excluded by the absence of spasms and of the severe pain in the back. In those cases not running a rapid course *acute poliomyelitis anterior* is excluded by the absence of wasting and by the normal state of the electrical reactions and the absence of fever.

Acute myelitis of the ascending variety shows always marked loss of sensation, a tendency towards trophic disturbances in the skin, and wasting of some muscles, with diminution in their faradic irritability.

Prognosis.—Though the outlook is always extremely grave, the disease has been known in several cases to end in recovery. Occasionally the fatal termination has not occurred till the expiration of several weeks. The earlier the bulbar symptoms appear, the darker becomes the prognosis.

Treatment.—Till the pathology of the condition is demonstrated, little can be expected from any known method of treatment. The ordinary remedies indicated in myelitis may be tried, as counter-irritation to the spine, absolute rest, &c.

PARALYSIS AGITANS

This is a chronic affection known also as Shaking Palsy and Parkinson's Disease, characterised by the presence of oscillating tremors, continued muscular rigidity, progressive weakness, and a peculiar gait.

Etiology.—The disease is only seen in those past forty. Very rarely has it been observed to commence before this age. Men are more frequently affected than women. The etiology is obscure, but a neurotic family-history is commonly obtainable, though a history of direct inheritance of the disease is rare. Prolonged grief or worry and long-continued mental

strain in some cases appear to be causative, whilst in others a sudden violent fit of emotional disturbance or a profound fright has appeared to inaugurate the affection.

Traumata, as head-injuries or serious wounds in any portion of the body, have been known to cause the disease. In one typical case, still under the writer's observation, the affection started soon after a severe incised wound of the wrist, the oscillating tremor commencing in the injured limb and gradually involving the muscles of the other extremities. Prolonged exposure to cold, syphilis, the infectious fevers, sexual excesses, and long-continued muscular exertion, have also been supposed to be etiological factors.

Symptoms.—The obvious feature in the symptom-complex is the *tremor*. In the majority of cases it is the first sign to appear. It usually begins in one of the extremities, commonly in the hand or fingers, from which it spreads, often in hemiplegic fashion, to the corresponding foot and leg before invading the limbs on the opposite side of the body.

It consists of a series of regular continued rhythmic oscillations, four to six per second, which are at first of such short range as to be sometimes liable to be overlooked. The tremors are caused by alternating contractions and relaxations in opposing muscles; thus in the hand the movements of flexion, extension, abduction, and adduction are continually being produced in the fingers. Often these are very strikingly seen in the thumb and forefinger, which are constantly performing the movements brought into play by the chemist in rolling or rounding a pill; whilst often at the same time the wrist-joint is being alternately pronated and supinated or flexed and extended, and the other fingers moved at their metacarpo-phalangeal articulations.

The most important feature in the tremor is its *continuance during rest*. Usually it is little influenced by active voluntary movement; though it may be stopped completely for a few seconds whilst the patient's hand grasps an object firmly, it begins again, often more vigorously, after the transient stop. It may sometimes be inhibited in a similar manner for a few seconds by a strong voluntary effort, when immediately afterwards it begins more actively than before.

Upon attempting to write, the tremor momentarily appears to lessen, but the zigzag lines and the imperfectly formed letters show that it is continuously present; in advanced conditions writing becomes impossible. Mental emotion exaggerates the oscillations, and if one limb be firmly seized by the physician so as mechanically to prevent the movements they at once become exaggerated in the other limbs.

Sleep silences the tremor, but very occasionally some oscillations may be observed in a portion of one limb. The writer has frequently satisfied himself that after profound

sleep, whether natural or artificially induced, the oscillations are generally more vigorous during the following forenoon. Usually all the extremities show the tremor, though very occasionally it may be confined to one arm or to the arm and leg on one side; it is very often more marked in the legs than in the arms. The head and neck muscles are but slightly affected; often they entirely escape, though the oscillations of the limbs may be mechanically communicated to the head. The lower jaw, tongue, and more rarely still the facial muscles may show tremors, but the abdominal muscles remain unaffected. Though the above distinctive features of the tremor of paralysis agitans are almost constantly present and unvarying, it must be remembered that in very rare cases the oscillations have been reported to occur upon movement or during mental excitement only. In rare cases the tremor may be entirely absent throughout, as in one otherwise typical case of the disease long under the writer's care, which was also seen by Sir William Gowers, the tremor never appeared, though the peculiar gait and all the other characteristic features of the symptom-complex developed fully before death.

Muscular rigidity is almost invariably present; and even when the tremor is entirely absent this is so characteristic that the diagnosis may be obvious. With the chronic rigidity is always associated more or less *muscular weakness*, though this latter never amounts to complete paralysis. The rigidity involves the neck-muscles and those of the vertebral column most severely, but the flexors of the limbs are also not infrequently involved, so that the arms are sometimes flexed at the elbows, and the fingers on the palm or only at the metacarpophalangeal joints with the last two digits extended. The rigidity can be overcome at first, but in the later stages, though it never possesses the true characters of spastic rigidity, it may be so marked as to prevent extension of the flexed joints, except to a very slight extent. It is not, however, increased by passive motion; actual shortening or contracture of muscles sometimes, however, occurs in the hands and feet.

The facial muscles show their rigidity by causing a stony, expressionless, or emotionless physiognomy, which is often highly characteristic; this is the reason of the close facial resemblance which many of the victims of paralysis agitans bear to one another. The mask-like expression is best marked in those cases where the tremor is absent, and it differs entirely from the physiognomy of double facial paralysis, which is of a flaccid type.

The most noticeable result of the muscular rigidity is the establishment of certain characteristic postures or positions which affect the *gait*. Thus the head is bent forwards, and the spine always is partially flexed so that the patient's body

is in a stooping attitude. On starting to walk he arises with considerable slowness and deliberation, but after he has walked a few paces his steps become shorter and his pace quickens to a trot. In extreme cases it is quite painful to watch him: with the head bent towards the ground and his shoulders forwards, he appears to be starting to run, and at each step the casual observer expects that he will fall upon his face. Should a strong light be directly behind him he appears to be running after his own shadow upon the ground. This peculiarity in the locomotion is usually described as the *festinating* gait, and it is highly characteristic, if not pathognomonic of the disease. Occasionally there is a tendency to walk backwards (*retropulsion*), and many patients can walk better backwards than forwards. Most muscular movements are executed more slowly than the normal, owing to the rigidity; and this is possibly the explanation of the festinating or hurrying gait, the patient being unable to bring the back-muscles into action with sufficient rapidity to check the stooping attitude which throws his centre of gravity forwards.

Muscular weakness may precede the rigidity; usually both symptoms are observed to occur and progress together, and they may be noticed to precede the tremor, though commonly the tremor appears first.

In the case above referred to, where the tremor never appeared, the patient's neck became so strongly flexed and rigid that his chin could not be separated from his sternum, and he was fed with the greatest difficulty. When he attempted to walk it seemed that each of his rapid short strides would land him on the pavement upon the top of his skull.

Speech is sometimes markedly altered, but it differs materially from that met with in disseminated sclerosis. It is monotonous and weak, the normal modulations are absent, and there is no disturbance of articulation like the scanning speech of sclerosis. When the patient attempts to speak he does so with very marked deliberation; but, like to what occurs in walking, the words gallop after each other, being sometimes shot out in an explosive manner which is quite characteristic.

The *reflexes* are normal, very occasionally they are increased, and sometimes even a false ankle-klonus may be elicited. The electrical reactions are normal and the muscles do not waste. The sphincters are intact; the writer has often noticed retention of urine, but it was always in patients who were taking hyoseine, except in one male case where the catheter had to be used often, but not constantly.

Sensory disturbances are slight; rarely is there any severe pain complained of except such as may be accounted for by the abnormal positions caused by the rigidity. A very common source of complaint is a distressing sensation of

burning heat in some parts of the body ; much less frequently do patients complain of cold. Sweating is often present, and this may be confined sometimes to an isolated region. Most victims of the disease cannot tolerate much bed-clothing at night, and, owing to the discomforts caused by the abnormal postures resulting from rigidity, the nursing of them in the advanced stages of the disease becomes a very wearying occupation.

Mental changes are sometimes observed. These occur oftener than most descriptions of the disease would imply. Thus, loss or a considerable impairment of memory in the writer's experience is a very common or almost constant feature in the later stages, as is almost always a more intense gloominess and constant depression of spirits than is witnessed in most chronic diseases. Occasionally dementia or other form of insanity develops before death.

Course, Duration, and Prognosis. — The disease is essentially chronic, and, though liable to occasional pauses in its advance, it has in the great majority of cases a more or less steadily progressive character. It must therefore be regarded as incurable.

All sorts and degrees of variations are observed in the rate at which the tremor passes from the limb first affected to the other parts. As a rule, the patient may be expected to become bed-ridden within a period of less than seven years from the appearance of the tremor, but shorter and longer periods are often noticed. Death is not due directly to the paralysis agitans ; but the miserable restlessness, broken sleep, sense of extreme fatigue, and general muscular weakness which supervene in the late stages of the disease produce eventually a worn-out condition of both mind and body, in which the most trivial bronchial or other ailment is quite sufficient to terminate the patient's frail tenure of life. Not infrequently the end comes in the form of an apoplectic seizure, entirely independent of the original disease.

Pathological Anatomy. — The changes which have recently been observed in the spinal cord by the modern methods of examination have rather tended to strengthen the old theory of the pathogeny of the disease as being of the nature of a premature or exaggerated senile degeneration. These consist of sclerotic changes in the vessels, perivascular sclerosis in the grey matter, and proliferation of the neuroglia in the lateral and posterior tracts. Probably similar senile changes will be found in the cerebral cortex. Some authorities, reasoning from the fact that the tremor stops during sleep and that it spreads usually from limb to limb in a hemiplegic fashion, consider that the disease is not of spinal origin, but that its seat must be found in the motor cells of the cerebral cortex. This view is strengthened by the observation that the supervention

of a hemiplegic attack silences the tremor on the affected side of the body.

Diagnosis.—The likelihood of an error in diagnosis only occurs in those cases where tremor never develops, but if the gait be observed in these examples of the disease it is quite characteristic ; moreover, muscular weakness and rigidity are invariably present.

Disseminated sclerosis resembles the affection, but only in a superficial way. Its tremor is equally characteristic. It is an *intention-tremor*, only produced when movement is attempted ; there are marked nystagmus, optic nerve-changes, and scanning speech ; rigidity causing alterations in posture and gait is absent, as is also the characteristic mask-like expression of the face, and this disease always occurs at a much earlier age than paralysis agitans.

General paralysis of the insane, with its tremor of the tongue, lips, and hands, can scarcely be mistaken for paralysis agitans, since the nature of the tremor is so different, never possessing regular rhythmic oscillations when the patient is at rest. The characteristic mental, speech, and paralytic symptoms and signs are pathognomonic of general paralysis.

Hemiplegia followed by tremor in the paralysed limbs is differentiated by the history of the suddenness of the original attack and by the fineness or shortness of the amplitude of the oscillations, which do not extend to the muscles of the unaffected side.

Senile tremor occurs in patients of a more advanced age. The head, which escapes or is but slightly affected in agitans, is the chief seat of the obvious shaking or trembling, which is much aggravated by, or may only be observable during, active movements. When this disease affects the limbs it always begins on both sides of the body at the same time, whilst agitans is usually hemiplegic in its onset.

Simple senile paraplegia may simulate the disease, as in one case seen by the writer, but the ultimate involvement of the sphincters and the extension of the weakness to the muscles of deglutition or speech may clear up the case. The predominant feature in this condition is a gradually increasing weakness of the legs, usually without marked tremor ; but an important point in the differential diagnosis is that, though the malady is a progressive one, it is liable to a degree of temporary improvement which is never observed in true paralysis agitans, and the characteristic attitudes of this latter disease are not present in senile paraplegia.

Treatment.—Rest of body and mind, obedience to all known health laws in the early stage, and careful nursing in the later stages, afford the best hopes of prolonging life and ameliorating the many discomforts and miseries of the disease. Of the innumerable drugs recommended with the view of

diminishing the tremors few have any decided action. Hyoscine is the only one likely to give satisfactory results; the hydrobromide should be commenced in doses not exceeding $\frac{1}{32}$ gr., and gradually increased to twice this amount. The writer, of late years, has satisfied himself that by the judicious use of this drug very great relief can be given to the restlessness, and marked diminution in the intensity of the oscillations may be produced for considerable portions of the waking hours. It may be combined with bromides, and occasionally with antipyrin. A prolonged course of arsenic, in combination with moderate doses of strychnine and valerianate of zinc, is sometimes beneficial when tonic treatment is indicated. Recently Clément has reported marked results from the administration of formic acid.

PARALYSIS, BULBAR

Under 'Muscular Atrophy of the Progressive Spinal Type' upon page 1065, is described a disease whose anatomical basis essentially consists of a chronic degeneration of the entire motor tract. Both the upper and lower segments of the tract are generally involved, but occasionally the degeneration only shows its presence clinically by the signs and symptoms arising from the involvement of the lower segment of the path. As already stated, the main clinical picture is that of a progressive wasting of the muscles, associated with some spasticity, the combinations of these two latter conditions constituting a number of varieties of the disease which are, however, strictly speaking, but different stages of the one degenerative process in the motor path.

Obviously the symptoms will vary as the degeneration happens to be most advanced in any particular region or section of the motor path. Thus, when the change begins in the bulbar nuclei the symptoms of paralysis of the tongue, lips, and pharynx may overshadow or dominate those observed in the limbs, which usually follow and become marked, should the patient survive. In other cases the bulbar symptoms appear later, being evidently secondary to the progressive muscular atrophy or amyotrophic lateral sclerosis (p. 1067). Though chronic or progressive bulbar paralysis has therefore no title to be regarded as a special entity or independent disease, for convenience its signs and symptoms are described under the following title:

PROGRESSIVE BULBAR PARALYSIS

This is also known as Progressive Glosso-pharyngo-labial, or Glosso-labio-laryngeal Paralysis.

Etiology.—The etiological factors supposed to be operative in progressive muscular atrophy are catalogued upon p. 1065; they are exposure to cold, trauma, and over-exertion of the muscles, mental depression, &c. Little or nothing of the real causation is, however, known, and heredity is only observed in an insignificant proportion of the cases, though a rare infantile type has been recorded. The most common period for the appearance of the affection is about the age of fifty or somewhat later.

Pathology and Pathological Anatomy.—Little need be said on this subject, since the pathological changes occurring in progressive muscular atrophy, of which this disease is but a part, have been fully described upon page 1068. The disease is a degenerative one, occurring not only in the spinal tracts, but in the nuclei situated in the medulla or bulb and in the pons. The ganglion-cells constituting the nuclei of the hypoglossal nerve are most profoundly degenerated; those of the spinal accessory and vagus are also considerably involved, and to a less extent those of the seventh nerve, the glosso-pharyngeal, and more rarely the motor nucleus of the fifth. The ganglion-cells of these nuclei lose their processes, and finally may themselves entirely vanish along with the fine network between them. Sometimes there is a marked increase in the interstitial connective-tissue elements. There is usually obvious atrophy of the fibres of the hypoglossal, accessory, and pneumogastric in the medulla, and of the extra-bulbar roots as well. The pyramidal tracts are also often involved and, as already stated, the spinal degeneration may be more or less complete. The affected muscles, with their intramuscular nerve-branches, show marked degenerative changes irregularly scattered throughout their mass. The changes are thus identical in nature with those found in progressive muscular atrophy. The lower motor segment going to the paralysed muscles suffers in some cases, whilst in those where wasting is absent the upper motor segment alone is involved, though usually the entire motor tract for the paralysed muscles is invaded by the degenerative process. It is considered probable that the nutritional changes are the result of some unknown toxic substance.

Symptoms.—The tongue being earliest and most deeply involved, *speech*-symptoms usually draw attention to the disease. At first fatigue in speaking is experienced by the patient, then indistinctness of articulation follows owing to a difficulty in the pronunciation of the linguals or linguodentals, as seen in the *l*, *r*, *d*, *t*, and *s* sounds. The labials cause trouble at a somewhat later period, *w*, *p*, *m*, *o*, and *u* being pronounced with difficulty; *b* is often sounded as *m*, and *p* as *v*, with a nasal intonation; and whistling is impossible. Soon the tongue is protruded only with difficulty, and finally

its tip can just be brought slowly and with effort to the margin of the lip; like the other muscles, it will be found usually wasted and wrinkled, but sometimes it is large and flabby. Ultimately the power of articulation entirely disappears.

Difficulties in *deglutition* usually succeed the speech-symptoms, but they may occasionally precede them. The food not being carried backwards by the tongue in the first stage of swallowing, and the palatal muscles being paralysed, liquids may regurgitate through the nose; hence pultaceous foods are less difficult and dangerous to swallow, especially as the laryngeal muscles are also weakened, and solid particles or liquids are liable to find their way into the larynx. All the pharyngeal muscles are ultimately weakened or paralysed, so that complete aphagia may ensue, and difficulty in mastication often is observable.

Laryngeal palsy is, however, seldom complete, though the voice is monotonous and low-pitched owing to the weakness of the muscles moving the vocal cords, but total aphonia seldom occurs. Cough is common from the escape of particles of food into the larynx, but it is always markedly feeble and accompanied by little sound.

Respiration ultimately becomes affected before death, usually from involvement of the trunk-muscles, or respiratory centre, or from aspiration-pneumonia.

The weakness of the muscles about the mouth shows itself early, as already stated, in the difficulty of pronouncing the labials; at a later stage the lower lip falls, and the mouth cannot be kept closed, so that the saliva is continually dribbling away and causing much discomfort. If at this advanced stage the patient's mouth be widely opened the tongue will be observed lying inert behind the teeth and incapable of lateral motion, though fibrillary twitchings may be observed in its substance; the velum will hang motionless, and the finger-tip may be carried over the palate, pharynx, and upper surface of the epiglottis without exciting the normal reflex contraction, though there is no loss of sensation in the mucous membrane, and taste is intact. The laryngoscopic examination at this stage will show the weakness of the adductors, the glottis remaining partially open during attempts at speaking.

The zygomatics and levator labii superioris, losing the opposition or antagonism of the lower labial muscles, cause the production of a deep and often characteristic furrow running downwards and outwards from the wing of the nose; the presence of this, with the open expressionless mouth and the dribbling of saliva, gives a pathognomonic physiognomy to the disease.

The paralysis is always bilateral and symmetrical, and is almost always accompanied by atrophy of the glossal, labial,

palatal, pharyngeal, and laryngeal muscles, and often by wasting of the jaw-muscles. The electrical reactions are but slightly influenced, only a very partial reaction of degeneration being obtained even in the late stages, owing to many of the muscular fibres remaining intact.

There is a type of the disease occasionally observed in which loss of reflex action and atrophy of the affected muscles do not occur, but a marked spastic condition is present in which there is great increase in the deep reflexes of the lower jaw and facial muscles. A tap on the chin in this type of the disease will produce a rapid jaw-jerk, and there may even be a distinct clonus on depressing the jaw; these cases correspond to the spastic type of paralysis seen in the limbs when the degeneration is mainly confined to the upper segment of the motor tract. The ocular muscles are never affected: sight, hearing, taste, smell, and tactile sensation are also always unaffected. The intellect usually remains intact, but there is almost constantly observed a state of unstable equilibrium in the emotional sphere, the patient being easily moved to tears or laughter, occasionally accompanied by an inspiratory stridor which accentuates the peculiarity of the physiognomy. The pulse is usually quickened considerably, and, as already stated, the signs of progressive muscular atrophy or amyotrophic lateral sclerosis are generally present in the limbs.

Course, Duration, and Prognosis.—The disease is an essentially progressive one, though short pauses in its progress may be occasionally observed. It is invariably fatal, and usually the end occurs within a period of two years from the first detection of the insidious onset, either from asphyxia caused by aspiration-pneumonia, or from paralysis of the respiration.

Diagnosis.—*Diphtheritic paralysis* may cause a symptom-complex simulating progressive bulbar paralysis; but the more speedy onset, the history of the infective stage, the absence of marked wasting of the muscles, and the fact that the palsy is mainly confined at first to the palate and afterwards usually affects the visual accommodation, and the early loss of the patellar reflex, are sufficient to prevent mistakes.

Syphilitic meningitis, caries of the occipital foramen, and tumours within the medulla or pressing upon it from without, may cause similar symptoms, but the paralysis is never so completely symmetrical in these cases, and always there are other parts than those supplied by the bulbar nuclei affected; headache, vomiting, and optic neuritis are likely to be present, one or other or all three together. In the diagnosis of progressive bulbar paralysis the look-out should always be in the direction of the spinal lesions (which usually ultimately appear) causing progressive muscular atrophy or amyotrophic spastic paralysis.

Pseudo-bulbar paralysis, about to be described, is never of insidious or slow onset; its differential diagnosis is detailed under its own heading.

Disseminated sclerosis cannot conceivably be so distributed as to produce medullary symptoms only confined to the motor nuclei.

Treatment.—Nothing will prevent the fatal issue, but careful feeding may prolong life considerably. Strychnine hypodermically, and galvanism of the medulla by passing a weak current from the one mastoid process to the other, have often appeared of some benefit in assisting the deglutition. Atropine or belladonna has been recommended to check the saliva, but the discomfort which it produces outweighs any benefit.

Progressive bulbar paralysis has been recorded of an *infantile hereditary type* occurring as a family-disease; the recorded cases are, however, very few.

A progressive *asthenic type* is occasionally met with in which there are no anatomical lesions discovered after death; as in the infantile form, the upper part of the face is usually also involved, and sometimes the ocular muscles. Oppenheim regards this type as constituting a distinct disease or an independent symptom-complex, having no connection with the degenerative variety. It is liable to remissions, and has a sub-acute onset. The cases of chronic bulbar paralysis occurring in young subjects which have been reported as cured probably belong to this category. The condition is supposed to be the result of a toxæmia, and this view is strengthened by the marked exhaustion of the affected muscles, which show no signs of atrophy or of the reaction of degeneration.

ACUTE BULBAR PARALYSIS

This condition, from the suddenness of its onset, has been called Apoplectic Bulbar Paralysis. The symptoms are implied by the name, and in the main correspond with those already described.

Etiology and Pathology.—The chief causes of the sudden paralysis are thrombosis leading to rapid softening in the medulla and pons, hæmorrhages into this portion of the nervous system, and, more rarely, embolic plugging of the basilar or vertebral arteries.

The thrombosis is usually the result of atheroma or arterio-sclerosis, the presence of which may also lead to bulbar injury in the case of falls or blows on the back of the head.

The pathological anatomy and pathogeny of acute bulbar paralysis are therefore practically identical with those of cerebral paralysis of the apoplectiform or hemiplegic character. There is, however, a distinct type of the affection which

commences as an acute *inflammation* of the grey matter constituting the motor nuclei of the bulb. This type corresponds to acute anterior poliomyelitis, and is sometimes described under the title of *acute polio-encephalitis inferior*; though usually occurring suddenly, its onset is sometimes sub-acute. Its etiology, pathological anatomy, and pathogeny are therefore akin to those of infantile paralysis. The attack often follows some infectious fever, and is met with chiefly in young subjects; it may be confined to one side of the body, constituting a lingual hemi-atrophy, alone or with facial paralysis or palsy of parts supplied by other cranial nerves.

Symptoms.—In acute bulbar paralysis there are rarely premonitory symptoms, but sometimes, as in thrombosis of the hemispheres, these may occur. The paralysis is sudden, sometimes happening with the abruptness and intensity of apoplexy, with total unconsciousness and convulsions; this is the rule in the hæmorrhagic cases, thrombosis more frequently causing a very transient loss of consciousness or severe attack of giddiness. The paralysis affects the muscles of deglutition, phonation, and mastication, as well as those in the lower facial zone, and sometimes those of the larynx, but it is rarely symmetrical, save in the acute hæmorrhagic form. Hemiplegia or paraplegia of either or both arms or legs is usually present, and all sorts of combinations may be observed in this respect. Thus an alternate hemiplegia may be seen in which the arm and leg are paralysed upon the side in which there is the least or no involvement of the muscles supplied by the bulbar or facial nerves. The paralysed muscles are rigid; this is observed sometimes at the onset by the firm contraction of the masseters. The deep reflexes are increased; but atrophy of the muscles rarely is noticeable, save in the tongue, because in the majority of cases the lesion is mainly *supra-nuclear*.

Respiration is usually seriously affected in severe cases, as seen in Cheyne-Stokes breathing, dyspnœa, and lividity; the temperature is generally elevated and the pulse is rapid. Sensory disturbances, as paræsthesia of one side or trigeminal anæsthesia, are observed, but the intellect is intact; in cases which survive, emotional instability becomes well marked.

Duration, Course, and Prognosis.—The attack is often rapidly fatal, but when the lesion is not caused by basilar thrombosis recovery may take place. After the initial onset, if the paralysis ceases to deepen, improvement, ending in recovery, may set in within a week and be complete inside a month; but if by the expiration of this latter period there be no marked improvement, the paralysis will be probably permanent. The ultimate prognosis in those cases not ending fatally within a few weeks will depend upon the nature of the underlying vascular lesion, being obviously bad where

advanced general arterio-sclerosis and renal changes are present, as a recurrent attack may be expected. Symptoms of extension of the mischief to the pons or to the corpora quadrigemina, as betrayed by nuclear ophthalmoplegia, deepen the gravity of the case.

Diagnosis.—The sudden onset, with the asymmetrical characters of the bulbar and other paralyses, is usually quite sufficient to distinguish the disease from the degenerative progressive type. *Pseudo-bulbar paralysis* must be differentiated. It rarely, if ever, follows a single apoplectic attack, the bulbar palsy only showing itself after a second seizure has involved the sound hemisphere; the motor weakness is slighter in degree, and the tongue does not waste; the larynx usually escapes, and reflex action is not diminished.

Treatment.—This resolves itself into the general management indicated in cerebral hæmorrhage and thrombosis. When the paralysis has ceased to deepen or improve, the main indication is that of judicious feeding; but even in the earlier stages the dangers of aspiration-pneumonia and asphyxia should be guarded against.

PSEUDO-BULBAR PARALYSIS

This is a rare condition in which bulbar palsy is due to a bilateral lesion situated in some part of the motor path in the cerebral hemispheres, the bulb being intact. Disease in the ascending frontal convolution on both sides, involving its lower part, or a lesion in the lenticular nucleus may produce all the symptoms of bulbar palsy, the lips, tongue, masseters, and pharynx being involved. A lesion limited to one side, though it may weaken the corresponding facial and tongue muscles, will not affect mastication or deglutition or interfere with the larynx.

The pathological lesions are almost certain to be the result of a widespread arterial sclerosis, though the possibility of a syphilitic origin must not be lost sight of. The symptoms are in one respect highly characteristic: they are abrupt and sudden in their first onset. The primary seizure is usually hemiplegic, from which more or less recovery may ensue; it is followed by another attack, which involves the opposite side of the body, after which symptoms of bulbar palsy first show themselves. In extremely rare cases the hemiplegia has been found only on one side, in which case the hypothesis has been advanced that, owing to some developmental anomaly of the innervation, the related functions of the two hemispheres are altered.

The paralysed muscles, including those of the tongue, do not waste, their electrical reactions are normal, and reflex action is preserved.

Owing to the underlying cause being a widespread one, the vascular supply of the brain through its sclerosed vessels eventuates in the production of multiple scattered degenerative changes, which usually show themselves in mental failure, often terminating in total dementia.

The paralysis of the bulbar nerves is seldom complete and never quite so symmetrical as in progressive bulbar degeneration, and the laryngeal muscles are much less likely to be involved. Oppenheim believes that unmixed pseudo-bulbar palsy is rare, most of the cases upon careful *post-mortem* examination revealing minute multiple changes in the pons and medulla, for which condition he prefers the term *cerebro-bulbar glosso-pharyngo-labial paralysis*. The respiration is often seriously interfered with in such cases, and Cheyne-Stokes breathing is a common symptom; sometimes optic, acoustic, and trigeminal sensory symptoms are also present.

An infantile type of pure pseudo-bulbar paralysis has been observed in association with cerebral infantile paralysis.

Diagnosis.—The chief points in the differentiation of the pseudo from the *acute apoplectiform* variety of bulbar paralysis have been mentioned upon page 1179; the main characteristic of the disease at present under consideration is the appearance of bulbar palsy only after a second or third distinct hemiplegic attack. From the *progressive chronic degenerative type* the diagnosis is usually easily made by attention to the abrupt hemiplegic onsets which always precede the bulbar symptoms, by the evidences of mental failure, by the absence of the true symmetrical distribution of the palsy, as well as by the occasional optic neuritis and other proofs of a multiple lesion.

Prognosis.—A fatal termination is the rule; but often this may be delayed for months or years, during which remissions may occur, usually followed, however, by a deepening of the mental deterioration.

Treatment.—The management of the case resolves itself into the selection of proper methods of feeding and nursing, and the exhibition of such agents as may be hoped to possess some influence over the underlying vascular condition.

Paralysis, Alcoholic, Arsenical, and Mercurial are described under 'Neuritis.' Lead Palsy is referred to under 'Lead-poisoning,' and Diphtheritic Paralysis is described on page 299.

PARALYSIS OF THE FACE

This is described under its own title upon page 414, and the palsies of other cranial and spinal nerves will be found under 'Paralysis, Peripheral.'

PARALYSIS, GENERAL, OF THE INSANE

This is a chronic degenerative malady characterised by progressive mental deterioration, speech-disturbances, and paralytic seizures, always associated with cortical cerebral atrophy and with spinal changes of a less definite character.

As seen by the above definition, general paralysis of the insane must be included in the category of organic cerebral affections, though also justly described as a *psychosis* under the title of Paralytic Dementia.

Etiology.—The disease is ten times more common in men than in women, and, although a few instances have been reported as occurring in early and late life, in the great majority of cases it makes its appearance between the thirtieth and fiftieth year, a large percentage occurring between thirty-five and forty.

Syphilis plays a most important rôle in the etiology of the disease. The proportion of cases in which an early syphilitic infection can be traced has been placed by some authorities at over 90 per cent. The unquestionably close relationship which exists between an antecedent attack of syphilis and general paralysis has been practically demonstrated by the remarkable inoculation-experiments of Krafft-Ebbing. After selecting eight general paralytics in whom no history of syphilis could be obtained, he inoculated each with the active virus from hard chancres and mucous patches. Though the experiment in each case was repeated from fifteen to twenty times and all the patients were kept under observation for almost five months, no symptom or sign of syphilis was produced, practically proving that in these cases a previous syphilitic infection had occurred. In this almost constant association of antecedent syphilis the disease shows a close relationship to locomotor ataxia, with which it is linked by other interesting bonds. This association, however, by no means proves that syphilis is the active etiological factor in either disease, since such a trifling percentage of syphilitic patients ever develop either *tabes* or general paralysis. It is in connection with this problem that the interesting theory and experiments of Edinger assume importance. Believing that the second factor in the production of these two allied diseases consisted of *an over-exertion of certain definite nerve-paths*, he was able to produce *tabes-like* lesions in the spinal cord of a rat which previously had been made anæmic by pyrocin, on compelling the animal to turn a treadmill for several weeks. Cases of *tabes* have been reported by Purves Stewart and others in which the ataxic symptoms have first appeared after some prolonged special muscular exertion.

If the probability of this theory be accepted, the great importance of the following etiological factors in general paralysis is easily conceived. The disease is one which almost always appears in those living a strenuous intellectual existence involving periods of prolonged mental strain, sustained worry, or protracted business anxieties; hence it is met with in speculators, keen business men, ambitious artists, literary men, politicians, &c. Alcoholic and sexual excesses undoubtedly play a considerable part, probably as a consequence of the constant emotional excitement following such indulgences; most of the general paralytics are married men, and the majority of them are gross feeders and individuals who give loose rein to all their appetites. The progressive tendency towards high pressure in the life of mercantile communities and the newer methods of juggling with financial projects are sufficient to explain the steady increase in the frequency of this formidable malady, as demonstrated by the statistics of each decade. It is always an extremely rare affection in rural communities. Heredity is a factor not of much consequence—it probably only acts as a predisposing cause; the cases occurring in childhood probably always are in the offspring of syphilitic parents. In a few recorded cases the exciting cause was a severe head-injury. There is little to support the contention of Ford Robertson that active bacterial infection from the intestinal and respiratory tracts is an etiological factor in the production of the symptoms; the bacterial invasion is probably the result and not the cause of the disease.

Symptoms.—These are most varied in their characters and groupings. In some cases the mental and in others the motor-spinal or the motor-cerebral symptoms predominate in the clinical picture, so that at least a dozen of so-called ‘types’ of the disease have been described; but all these eventually meet at the common goal of total dementia unless the victim’s life be cut short by some complication.

The disease has been divided into several stages, but the symptoms supposed to be peculiar to a particular stage may appear at any period during the progress of the malady, so that little clinical aid is obtainable from a study of the disease from this point of view. Nevertheless, the enormous importance of the earliest possible recognition of the disease justifies a careful look-out for and analysis of the symptoms or warnings which are most likely to herald the advance of the malady.

Prodromata.—These may be entirely absent in the rare acute form of the disease, which with explosive suddenness is ushered in by symptoms of acute delirious mania or by an epileptic fit. The finer mental changes are liable to escape detection, being only visible at first to those in close relation-

ship with the individual. Some slight change in character, which is often of the nature of an exaggeration of some peculiarity present in the individual from childhood, may be the first evidence that he is not 'what he was before.' Thus, if he has been always quick in his temper, he now shows unusual irritability from causes which formerly failed to ruffle him. His power of concentration is weakened and some feebleness may be noticed in his will; he ceases to focus his attention upon the mental exercise before him, and does not apparently recognise his failure. His affections may undergo some change, and moral perversions of various kinds may be noticed by his performance of acts which he formerly regarded as discourteous, dishonest, or indecent in others. Such acts may be first observed to occur after a dose of alcohol which formerly had no effect upon his mental equilibrium. These mental prodromata deepen into the symptoms of the established disease, any of which latter may also appear as the first warning to his immediate friends; thus his premonitory symptoms may be those of an abnormal idea of his own importance, wealth, and station, or a carelessness, forgetfulness of, and indifference to business. Motor symptoms, as restlessness, slight tremors of the lips and hands, difficulty in articulation or in letter-writing, may precede the mental warnings; pupillary rigidity or an unsteadiness in his gait may be the first sign that anything is wrong. Occasionally sensory disturbances are complained of, such as transient diminution in the acuity of vision, hearing, and sensation; tinnitus, vertigo, or even passing illusions or hallucinations of the senses may be experienced. If he be an artist or musician, at this stage a close observer may perceive that some ill-defined signs of deterioration may be made out in his work.

Very often the above warnings only come to the physician's knowledge after the invasion has been so decided and advanced as to first arouse the alarm of the patient's friends, in which case they are elicited only on careful cross-examination; but they are of great diagnostic value in revealing the true nature of the insidious onset, as they may precede the definite onset of the malady by several months.

Mental Symptoms of the Established Disease.—These are exaggerations of the warning symptoms: the character of the patient becomes rapidly changed, he gets heartless and careless about his family and home duties, neglects his business, though his mind may be filled with schemes for making colossal fortunes, and he may take to the spending of money lavishly, and may at the same time show very little scrupulosity in the methods for obtaining more. His will-power is markedly weakened. His memory fails early, especially for recent events; he becomes unable to calculate or tot figures, and may

soon cease to have a right conception of the day of the week or month, or even to remember the number of the year in which he is living. At this stage restlessness is more common than apathy, and there is a liability to violent outbursts of anger and jealousy, or to the performance of some act violating all the laws of decency.

These mental changes may precede any motor signs for many months; but, on the other hand, some cases occur in which marked motor disturbances exist for long periods without any obvious change being detectable in the patient's mind or conduct.

As the mental deterioration increases at a steady or a jerky pace towards dementia, *delusions* are common. The most characteristic type of these is the 'grandiose.' The patient fancies himself to be a king, a multi-millionaire, a great discoverer, or the possessor of boundless influence, power, or knowledge; not very infrequently his disordered imagination presumes to impersonate the Deity. One day he is an emperor, and the next he may be a statesman, orator, or artist. In some rare cases this expansive delirium or mental exaltation never shows itself, being replaced by the opposite condition of deep melancholia or hypochondriasis; and even where the exaltation is to become a prominent feature it is very often preceded by some temporary depression. The great majority of all the varied types of the disease exhibit this form, in which the mania is associated with grandiose delusions.

Delusions, mental exaltation, depression, or excitement, and the usual symptoms of mania may all be wanting from the start, the patient passing steadily into a state of stolid stupidity, ending in simple dementia, the weakness of mind in this demented type being the only progressive mental change. Such patients often have epileptiform convulsions, and tend towards becoming obese; but, as Goodall points out, there is at any moment a danger of the most docile of them becoming violently maniacal. Though all cases proceed finally towards complete dementia, it will be observed from the above remarks that the insanity which accompanies general paralysis may take the form of delusional insanity, acute delirious mania, hypochondriasis, melancholia, or simple dementia, or two or more of these forms may be grouped together in the same case.

The mental condition is peculiar in a type of general paralysis which shows marked tendency towards remissions. The disease somewhat suddenly sets in with the usual almost typical grandiose delusions and evidences of unbounded benevolence, but after some months complete recovery ensues. Following some excitement or strain, a few years later another attack occurs, this time the mental condition being one perhaps of only extreme restlessness, with insomnia,

or more probably of considerable depression, recovery again following. Such cases constitute the *circular* form of the disease; but after the lapse of several years they all finally die of general paralysis.

Motor Symptoms.—In the above description of the various mental changes met with in the disease, from the prodromata to the final dementia, the mention of motor symptoms is omitted for the sake of clearness. These, as already stated, may first show themselves as warnings before the real invasion-period. The facial muscles show early the serious nature of the disease in many cases, so that the condition can be recognised at a glance. The face is flabby and expressionless or gives one the idea of a profound stolidity, but upon the patient commencing to speak a marked tremulous condition of the muscles reveals itself, showing a loss of control. The lips and tongue are the seat of marked tremor; the tongue is projected with considerable difficulty by a series of ataxic jerkings, and fibrillary twitchings may be observed in it. At a later stage it is paralysed and cannot be protruded beyond the teeth. Speech is characteristically affected: it is halting, slow, and drawling, and some syllables are slurred over or clipped out. Words of many syllables, which are pronounced with some slight difficulty by normal individuals when in a hurry, cause the general paralytic to stumble or fall completely in his speech—*i.e.* words like ‘arithmetical,’ ‘protuberance,’ &c., are bungled by his quaking tongue and tremulous lips. The prominence of the cheeks is sometimes noteworthy, the patient appearing as if he still possessed his infantile sucking-pads, and this idea is strengthened by the dribbling of the saliva and by the ceaseless motions of the mouth-muscles, which continue sometimes to execute the movements of sucking.

The *handwriting* changes. The letters are unevenly formed, the strokes showing shakiness and unequal degrees of length and thickness: but the most characteristic feature is the omission of letters, of syllables, and sometimes of complete words. The letters, though correct, are frequently not connected with each other into words. In advanced cases words and letters may be hopelessly jumbled up, and his epistle may be absolutely unintelligible. The tremor sometimes shows itself in the muscles of the limbs; it is most varying in its fineness or coarseness, and, though best seen during the execution of movements, it also occurs during rest.

Pupillary rigidity is marked in more than half the cases, and may be the earliest of all the warnings. The pupils may not only be very sluggish or fail entirely to respond to light, but they are often unequal, irregular, contracted as in tabes, or occasionally markedly dilated. The knee-jerks show all varieties of change: they may be absent, sluggish,

exaggerated, or normal, or present on one side and absent on the other. The movements of the limbs may be executed in an abnormal manner; as seen in the changes in the handwriting, the finer movements of the fingers may be blurred or prevented, silencing the violinist or pianoforte-player and terminating the career of the artist. Other coarser motions are executed with awkwardness, the cause being probably an ataxic one combined with some paresis. The loss of power is usually not great at the early or even middle stage of the disease, which is therefore sometimes styled general 'paresis.' The gait becomes unsteady and may present all the characteristics of the typical tabes-gait; at other times it is highly spastic, the lateral tracts, or these with the posterior spinal tracts, being involved; the deep reflexes are exaggerated. The combination of tabes and general paralysis is a by no means rare one, and adds considerable interest to those cases where ataxia, Argyll-Robertson pupils, optic atrophy, Westphal's and Romberg's signs, lightning-pains, and bladder-symptoms have preceded the onset of mania.

A characteristic feature of general paralysis is the liability to paralytic seizures. One of these may, like a bolt from the blue, usher in the disease, the patient falling down in a severe apoplectic or epileptic fit, just as a fit of acute delirious mania may occur in a similar manner without warning, or as some flagrant outrage on decency or terrible crime may be the first indication of the disease.

The important feature, however, in the paralytic phenomena (once the disease has become firmly established) is the very frequent *recurrences* of apoplectic or epileptic attacks of short duration.

The patient is usually left hemiplegic, monoplegic, or aphasic after such attacks, which are usually ushered in by a more or less complete degree of unconsciousness, and are often accompanied by fever.

The paralytic symptoms pass off, as a rule, in a few hours or days, but each subsequent attack leaves a deepening of the mental weakness and of the muscular feebleness, as seen in the increase of the speech-difficulties and the impairment of the gait. The epileptic seizures may be of the nature of minor attacks in which consciousness is not obliterated, or they may show themselves as typical attacks of Jacksonian epilepsy. There is commonly an aura, usually of the nature of a visual disturbance; the convulsions may be confined to one side or pass over, as in true epilepsy, to the other side and become general. A true *status epilepticus* may supervene when the fits are numerous or frequent, and death may occur in this way. Occasionally motor symptoms may be absent in these attacks, which then may be purely psychic in nature, as seen in frequently repeated fits of brief excitement

or gloom, mixed up with or alternating with attacks of vertigo, aphasia, or total brief mental blankness.

Sensory symptoms such as those noted as warnings may continue: thus the fleeting loss of sight may progress to total, but much more frequently to only partial, blindness; and similar results may occur as regards the senses of taste and smell and common sensibility, which latter may almost entirely disappear. Illusions and hallucinations of the special senses are common, and in some cases the first symptom may be an evidence of this sort, the patient hearing voices or seeing images of strange faces long before any paralytic symptoms have appeared.

A further link between the disease and tabes may be sometimes observed in the appearance of trophic changes, as perforating ulcers, bed-sores, Charcot's joint-phenomena, or spontaneous fracture of bones; this latter is especially liable to happen, as the bones become very brittle in general paralysis. The association between tabes and general paralysis is best seen in the so-called '*spinal*' type of this latter disease; thus general paralysis has often been found to supervene upon a typical case of locomotor ataxia which has for many years exhibited spinal symptoms only. But the spinal type is not necessarily always of tabetic origin; sometimes general paralysis, with its typical maniacal features and delusions, supervenes upon a case of spastic paraplegia which had commenced and existed for a long time without showing any mental symptoms.

These cases showing a combination of the symptoms of tabes and general paralysis are often classed in a group under the name *Tabo-paralysis*, the main feature in their clinical picture being the frequency with which the primary tabetic symptoms are heralded in with atrophy of the disc. Mott and others regard the essential features of tabes and general paralysis as identical, in the one case the disease attacking the spinal cord and in the other the cerebral centres suffer; hence we sometimes find the term '*cerebral tabes*' applied to the disease under consideration—an unfortunate term liable to be confused with the totally different condition recognised as '*cranio-tabes*' which occurs in rickets.

Course, Duration, and Prognosis.—Though the disease usually has an insidious onset, it may commence, as already stated, in an explosive manner with acute delirious mania and terminate life within a couple of months. The general average of cases run their progressive course towards total dementia and death within a period of about three years. Remissions, however, are often met with, but in the cases where recovery follows soon after the initial invasion there is always a doubt about the accuracy of the diagnosis. The disease must be regarded as incurable, and practically the

gravest prognosis is always justifiable. Many cases, especially of the type met with in rural asylums or workhouses, may run a course of many years, showing remissions or halts of long duration. Often marked emaciation sets in early, but in the stage of dementia the patient becomes flabby and fat, and finally again wastes. In the intervals between the attacks in the circular form of the disease the symptoms entirely disappear, but the inevitable occurs, though it may be postponed for many years; no prognosis can safely be made in these cases as regards duration.

Ultimately with the progress towards complete dementia the paralytic symptoms or paresis deepen, so that the patient becomes bed-ridden and is unable to articulate, his sphincters fail, bed-sores form, and accidental contusions cause large hæmatomatous tumours. Cheyne-Stokes breathing often is observable, and the final issue may be the result of aspiration-pneumonia or asphyxia from impaction of food in the air-passages; he may succumb from some trivial intercurrent affection, from cystitis, diarrhœa, or general inanition.

Morbid Anatomy.—The changes are widespread and numerous. Though by some authorities they are still regarded as not constituting an anatomical basis peculiar to the disease, nevertheless the constancy with which atrophy of the cerebral cortex is found after death renders any hypothesis untenable which fails to recognise this morbid condition as the chief pathological feature in general paralysis.

The membranes are thickened, the dura especially, which may be adherent to the skull-cap along the vertex, and its inner layer is the seat of hæmorrhagic pachymeningeal changes; the opaque pia when pulled off brings usually some of the adherent cortex with it, but in long-standing cases it is noteworthy that this latter change is very unusual. Fluid fills the sulci and occupies the space formerly filled by the now wasted convolutions. The ventricles are dilated, their cavities distended with turbid fluid, and their ependymal lining is markedly granular, the fourth ventricle being always most deeply involved in this latter respect.

The brain is wasted and shrunken, the sulci are deepened, and the gyri are small, the convolutions of the parietal and frontal lobes suffering most. The wasting in other parts is also obvious to the naked eye, as seen in the vicinity of the island of Reil, the temporal lobe, the pons and medulla, internal capsule, and even in the basal ganglia to a slighter degree. The entire brain-weight is markedly diminished: it may fall from nearly 50 oz. to under 35 oz.; though there is great diminution in the white matter, the grey cortex is, however, always affected first, and is markedly withered in long-standing cases. The organ is much softer than the normal, and not infrequently foci of softening and of minute

hæmorrhages are studded throughout its substance. These may be visible to the naked eye in the cortex in recent cases, and may cause it to present a patchy congested appearance. Microscopically, the most important change demonstrated by the more recent methods of examination is an atrophic degeneration of the fine tangential fibres and ganglion-cells in the cortex. The interstitial fibrous tissue is increased, which is possibly a primary change; the vessels are sclerosed, the lymph-spaces dilated, and large spider or lymph connective cells are numerous. The optic nerve and the nuclei of the other cranial nerves are frequently also degenerated. In more than one-third of the cases there are marked changes of a degenerative character in the cord, chiefly in the posterior columns and in the crossed pyramidal tracts. The peripheral nerves are also found to show degenerative changes of the same general type as those seen in the cortex—viz. an increase in the connective tissue and wasting of the nerve-elements.

The pathogeny of the disease is still a disputed point, the problem awaiting solution being whether the disease should be regarded as a primary inflammatory process starting in the vessel-walls and interstitial tissue, or as a primary degenerative parenchymatous change starting in the nerve-elements. The general pathological picture is that which might be expected to be produced by the destructive action of some toxic agent, as held by Mott and Savage. The view of Ford Robertson that the toxæmia is of the nature of an auto-intoxication from the intestinal and respiratory tracts is much less likely to be correct than the hypothesis that the syphilitic toxin probably present at a remote period in every case has so diminished the natural vitality or resistance of the entire nervous system that certain tracts being exposed to '*over-exertion*' their nerve-elements succumb to degeneration. This view is supported by the fact that the disease is always more advanced, even in the early acute cases, in the cortex of the frontal lobes; and if the general etiological factor of over-pressure in intellectual pursuits be considered in conjunction with this, a reasonable working hypothesis results.

Diagnosis.—Various forms of *insanity* may easily be mistaken for general paralysis. It is necessary to bear well in mind that every form of mental unsoundness may be associated with the paresis in this disease; hence the problem of the differential diagnosis mainly consists in the exclusion of general paralysis by carefully noting the absence of such symptoms and signs as tremor of the tongue and facial muscles, Argyll-Robertson pupil, the speech and gait, and the ataxic characteristics often present. The peculiarities of the mania itself must not be relied upon too implicitly, since the exaltation observed in general paralysis is not confined to

this disease; and, moreover, the general paralytic is often at times markedly hypochondriacal, being depressed especially about the state of the patency of his bowels. The exaltation of general paralysis is, however, almost invariably of a benevolent type; the mental deterioration shows itself in the loss of will-power and in the establishment of the so-called '*facility*' which enables the general paralytic to be easily led and managed. The highest and most lately acquired mental accomplishments are those which are first lost.

The acute mania of general paralysis lasts usually a much shorter time than *simple acute mania*; *melancholia* may be differentiated often when occurring at the paralytic age by a history of previous attacks at an earlier period in the patient's life; *senile dementia* only appears after the age of sixty.

Alcoholic Dementia more closely simulates the disease than does any other form of psychosis, because in it, in addition to the mental symptoms, there may be tremor and speech-disturbances and great restlessness, combined with motor weakness. A careful examination of this latter sign will often throw light upon the difficulty, the paresis being traceable to a neuritis of the peripheral type, with its own characteristic distribution. The mental unsoundness, if it take the form of delirium tremens, is easily diagnosed; and the polyneuritic psychosis of Korsakow, which often occurs in chronic alcoholism with neuritis, is very remarkable. The patient, though bed-ridden for months, will relate with striking minuteness and vividness the details of a long walk which he fancies he has performed the previous day, describing the shop-windows and their contents and the people with whom he conversed in the streets during his ramble. *Lead-poisoning* is often accompanied by a mental state which, taken in combination with the palsy, may be mistaken for general paralysis; but the recognition of the peculiar selective action of the metal upon the extensor muscles, the colic, and the blue line along the gums, are characteristic.

Neurasthenia resembles the earlier stages of the disease closely, but the absence of eye-symptoms, tremor, the state of the knee-jerks, &c., and the study of the character-changes will exclude it. It has been held that the same causes which produce neurasthenia in the non-syphilitic determine the advent of general paralysis in those who previously suffered from syphilis, since the etiological history of both conditions is identical, if we exclude the history of the old syphilitic infection. The clearness and persistency with which the neurasthenic patient watches his special symptoms and describes his sensations is in marked contrast to the apathy and daily variations of the general paralytic, who, if hypochondriacal, will complain of one symptom to-day and of

another to-morrow. *Hysteria* occurring in men after middle-life should be regarded with grave suspicion; though the condition is very rare, most of such cases develop ultimately into general paralysis, and a close out-look should be maintained for the appearance of motor symptoms.

Cerebral Syphilis may exist with a train of symptoms closely resembling general paralysis. Though this latter disease need not necessarily ever supervene, the prognosis being very different in the two affections, close attention should be given to the diagnosis. In cerebral syphilis the paralytic phenomena are usually manifested in the loss of power over the muscles of the eyeballs, headache is often intense and localised tenderness of the scalp is present, and there may be optic neuritis. The diagnosis may be very difficult when a syphilitic *meningitis* involves the speech-centres and motor area, causing speech-disturbance and palsy of the limbs; but the irritative nature of the lesions is more prone to show itself in the form of a true Jacksonian epilepsy, in which more or less palsy remains between the seizures, proving the focal character of the affection. The paroxysmal mental seizures present in cerebral syphilis are of the nature of simple brief attacks of stupor or of unconsciousness of some hours' or days' duration, which usually clear up completely, in contrast to the marked progressive march of the dementia seen in general paralysis. Cerebral syphilis is, moreover, to a considerable extent amenable to active treatment by iodides in large doses. Only by close watching over a considerable period can extensive syphilitic arterio-sclerosis of the brain be differentiated.

Cerebral Tumour, whether syphilitic, malignant, or otherwise, must be differentiated by the focal character of its signs, taken in conjunction with the general clinical symptoms, as severe persistent headache, obstinate vomiting, and double optic neuritis.

Locomotor Ataxia, as already stated, not infrequently precedes the advent of general paralysis, and sometimes in a typical case of the latter disease all the signs and symptoms of ataxia may ultimately make their appearance. The differential diagnosis is easily made by noting the presence or absence of the symptoms peculiar to each affection. If the case be one of so-called *Tabo-paralysis*, the diagnosis depends upon the recognition of the combination of the symptoms of both diseases.

Epilepsy is not likely to be mistaken for general paralysis, but the physician should always suspect the advent of general paralysis in every case where epileptic convulsions make their first appearance in a male of forty years or upwards. After an ordinary epileptic attack there should be no signs of paralysis observable when the patient has awakened from

the slumber following his fit. In general paralysis marked paresis may remain for many hours or for a few days after each seizure. *Apoplectic attacks*, followed by hemiplegia, which slowly passes off after some months, can only be mistaken for general paralysis when they recur and are associated with considerable mental impairment; but the type of this mental failure always differs markedly from that seen in the general paralytic. The underlying arterio-sclerosis will always betray its presence in such cases by focalising symptoms, often of the bulbar-paralysis variety.

Disseminated sclerosis occurs, as a rule, in young subjects, and is only likely to be mistaken for general paralysis in those rare cases where the sclerosis is first ushered in, at a later stage, by an acute encephalitis, with focal symptoms, or after an epileptic or apoplectic seizure. The subsequent development of the scanning speech, nystagmus, and intention-tremor are, however, not to be mistaken for the speech-disturbances, paresis, and facial quiverings of general paralysis.

The examination of the blood in general paralysis may assist the diagnosis in doubtful cases. There is always a more or less progressive anæmia, as shown by a diminution in the hæmoglobin, with a leucocytosis caused by marked increase in the polymorphonuclear cells. The examination of the fluid obtained by lumbar puncture shows a considerable amount of albumin, accompanied by leucocytosis, especially marked after the paralytic attacks.

Prognosis.—This has been already discussed on page 1187; the disease should be regarded as incurable. Some authorities consider the prognosis as less grave if the advent of the disease has appeared within a period of seven years from the antecedent syphilitic infection; but cases of this kind recovering under antisyphilitic treatment should probably be regarded as examples of cerebral syphilis, and not of paralytic dementia.

Treatment.—No known drug possesses any power over the progressive march of this fatal malady. The early removal of the patient to a proper asylum is absolutely necessary in order to protect himself and his family from the serious consequences or ruin which may follow the perpetration of some crime or the vagaries of his grandiose delusions. At a later stage, when the paralysis and dementia have progressed to the degree which renders him incapable of doing harm to himself and others, he may be permitted to spend the remaining months of his life amongst his relatives, if their means will enable them to secure the services of highly trained nurses. The absolute rest of a quiet and well-regulated asylum certainly tends to check the rapidity of the progress of the malady by diminishing the excitement which his restless delirium is constantly keeping up. In every case, especi-

ally if the symptoms follow within a limited number of years after the syphilitic infection, the routine of an antisymphilitic treatment may be carried out as soon as the patient has been placed under restraint ; though little hope can be held out that a permanent benefit will result thereby. The plan of trephining and establishing drainage, with the view of permanently keeping the intracranial pressure at a low tension, has not been followed by such results as justify the procedure. Symptoms are to be treated as they arise ; thus hydropathy and the persevering use of the ice-cap diminish the tendency to paralytic seizures and often aid insomnia and appetite.

PARALYSIS, GLOSSOLABIAL.

(See under PARALYSIS, BULBAR, p. 1173.)

PARALYSIS, HYSTERICAL.

(See under HYSTERIA, p. 684.)

PARALYSIS, INFANTILE

The name is an unfortunate one in this respect, that many types of paralysis occur in childhood or infancy, some of these being of cerebral, others of spinal, and some of muscular origin. The disease under consideration is, however, known by the following synonyms, the use of any of which will prevent possible confusion—*i.e.* Poliomyelitis Anterior Acuta, Acute Atrophic Spinal Paralysis, Essential Paralysis of Children.

It is an affection occurring during the first few years of life, characterised by the presence of a transient fever, a more or less sudden loss of voluntary power in certain muscles or muscle-groups, which is soon followed by wasting of some of the affected muscles and by more or less recovery in others.

Etiology.—The disease appears somewhat more frequently in male children. Half of all the cases occur before the termination of the third year of life ; it is, however, rare before the first half-year and after the sixth year, though adults occasionally suffer. Its cause is supposed to be of infective origin, and this view is strengthened by its occasional appearance in a well-marked *epidemic* form. The nature of the infection is unknown ; similar symptoms have been produced in animals by the introduction of various micro-organisms, and the affection has been observed to follow measles, influenza, whooping-cough, and other infectious

diseases. Cold, trauma, and over-exertion were formerly believed to be etiological factors. It occurs much more frequently in warm or hot weather.

Symptoms.—In typical cases which have been closely observed the disease begins suddenly, like an acute infectious fever, with malaise, vomiting, and increase of body-temperature, which may reach 103° or even 104° . Occasionally stupor, delirium, and convulsions have been noticed; but these, with the fever, are of such very transient duration that in many cases the first symptom noticed is that on awaking the child is observed to have lost the power of an arm or leg, or of both upper or both lower extremities, or of one arm and one leg on the opposite or, more rarely, on the same side of the body. Often there is marked tenderness or pain in the paralysed member or in the upper region of the spine. The presence of this pain may be the first thing which draws attention to the paralysis. Very seldom is even a trace of diminution of the sensibility of the skin to be detected.

The loss of power may be complete within twenty-four hours; not rarely it is complete as soon as it has been discovered. In its fully developed stage the paralysis is always a flaccid one, so that by the end of a week some wasting may be detected and the *reaction of degeneration* can be demonstrated. The irritability of the motor nerves is found to be decreased and is soon lost. The muscles do not respond to the faradic current, but they react to galvanism more markedly than in health; though the contractions are sluggish, they may be induced by a current which would be inoperative for normal muscle. In rare and very severe cases the muscles rapidly lose all irritability to both forms of current.

In testing the muscular irritability, even at this early stage (within a week) important observations may be made; thus, though the entire limb may be apparently paralysed, only one or more groups of muscles may be found in which there is complete loss of faradic irritability. These will remain permanently paralysed and wasted, whilst the muscles which have still retained their faradic irritability will probably eventually entirely recover.

Gradually some of the paralysed muscles begin to show signs of returning power, and after a few weeks or months it may often be found that all the muscle-groups are capable of acting under the power of the will, save a few. Thus in the arm the deltoid alone may remain permanently wasted and powerless; more frequently the biceps and brachialis suffer with it. In the leg the extensors of the feet and toes may alone be permanently damaged; they are usually more severely involved than the calf-muscles, so that talipes equinus is more common than valgus or varus. All sorts of combinations of individual muscles or muscle-groups may escape or

be permanently injured. Very rarely does complete muscular power return in all the affected groups. The legs are much more frequently the seat of permanent damage than are the arms, and the left leg is damaged oftener than the right. Sometimes the permanent paralysis may involve the entire limb, or even remain complete in one leg and in one arm or in both legs.

With the initial paralysis of motion there is, as already stated, usually no diminution in the acuteness of sensation; the early pain usually is transient, unless when neuritis is present, when it may be severe and lasting. The reflexes are often abolished completely in the affected parts, but return when the permanent improvement is widespread. Still states that the knee-jerks are not unfrequently present throughout. The sphincters are intact, except in the very rare cases where sensation suffers from grave involvement of the lumbar cord. The mental condition remains unaffected and bed-sores never form.

It must be remembered that in fat children atrophy may be entirely overlooked, but the reaction of degeneration will in such cases clear up any doubt if the marked flaccidity of the affected limb be not sufficient to indicate the specific nature of the palsy; muscles, however, which are merely weakened, but not wasted, will not give the reaction of degeneration.

The diminution in the response to faradism begins to improve after about four to six months in the muscles which are not hopelessly degenerated and atrophied; response is obtained, however, in muscles which are very markedly paretic owing to certain strands of fibres escaping or completely recovering. In the muscles which become early severely wasted the faradic irritability never returns, and usually within a year all trace of galvanic response also becomes invisible, unless electrical stimulation has been continuously kept up; but even then it ultimately disappears.

Grave deformities ultimately ensue in all severe cases; thus the arm, forearm, thigh, or leg may be so withered as to present the appearance of a bone only covered by skin. In patients affected at the usual early age the growth of the bones is checked, so that the long bones of the paralysed limbs remain much shorter than the corresponding ones on the sound side. The unaffected muscles, losing their normal antagonism, shorten, and serious contractures occur; structural alterations ensue in them so that they cannot be extended or flexed by forcible passive movement. The weight of the foot plays a considerable part in the deformities seen in the lower extremities, where these changes result in the production of the various forms of club-foot, already referred to, of which talipes calcaneus from paralysis of the calf is the least

frequent. The claw-hand attached to the fin-like withered upper extremity is produced in the same manner.

The joints become loose from the absence of the tension and support of the tendons passing over them, so that dislocations occur, the head of the humerus dropping downwards from the glenoid cavity and the head of the femur slipping out of the acetabulum. Though the trunk-muscles are seldom severely paralysed, spinal deformities, as scoliosis and lordosis, are not rare.

The affected limbs are cold, and often livid from deficient circulation of blood caused by vaso-motor disturbances.

Death is extremely rare as the direct result of the disease, save in severe acute attacks where the paralysis is general and involves the diaphragm; but in a small percentage of cases degenerative changes may long afterwards set in in the lateral columns, or even progressive muscular atrophy may develop after adult-life has been reached.

Pathological Anatomy.—The essential changes found after death in recent cases are those characteristic of an acute interstitial inflammation of the grey matter in the anterior cornua of the cord. The vessels are distended with blood, minute extravasations are seen in the horns, which are softened, the nerve-fibres and neuroglia show considerable swelling, the ganglion-cells are also swollen and granular, with their nuclei indistinct and their processes wasted. Numerous granule-corpuscles and leucocytes are found in the deeply congested grey matter; at a slightly later stage the ganglion-cells have almost entirely disappeared, and acute changes are found in the anterior nerve-roots. The pathological picture is thus one of a diffuse hæmorrhagic myelitis confined to the anterior cornua, but occasionally the adjacent lateral column and the meninges may be invaded to a slight extent. According to Marie, the diseased action begins as a thrombotic process caused by infective emboli lodged in the commissural branch of the anterior spinal artery.

The pathological changes observed in cases of long standing are also unmistakable even to the naked eye, the anterior horns being, in the site corresponding to the level of the affected muscles, distinctly wasted or shrunken, and the withering process involves the anterior nerve-roots. In cases of very long-standing extensive paralysis one-half of the cord is smaller than the other at the affected level.

Under the microscope the ganglion-cells are found to have withered up, a few only remaining; and the axis-cylinders of the nerve-fibres are absent, as traced through the anterior roots into the nerve-trunks. The interstitial connective tissue is increased; as a rule, the above changes are most frequently met with in some parts of the cervical or of the lumbar enlargement.

The changes in the muscles are of the usual degenerate type which follows upon destruction of the lower motor neurons: whole muscles or portions of them may be entirely replaced by fibrous tissue and fat. As a rule, normal strands of striated tissue may be discovered scattered throughout the mass of the degenerated muscle, some of the fibres being even larger than the natural in the vain attempt at hypertrophy. In some cases evidences of inflammatory action, followed by degeneration, have been discovered in the motor nuclei of the medulla. In most of the long-standing examples of the disease some degeneration may be observed in the neighbouring white columns.

Diagnosis.—Inflammatory conditions of *joints*, especially those observed in *acute rheumatism*, may closely simulate the disease in the stage of onset, and many cases are at first supposed to be of the nature of an acute rheumatic attack till the paralysis and wasting reveal their true nature. A careful examination will, however, prove that the loss of mobility is not arthritic; on attempting passive movements in joint-diseases the muscles resist in order to prevent the pain which is chiefly caused by moving the joint. In infantile paralysis, when pain is a marked feature its spontaneous character is usually easily recognised. When the disease occurs in adults, disordered sensation, as tingling or formication, usually accompanies the loss of power. *Rickets* and *Morbus Coxæ* are differentiated in a similar manner, and the history of the onset is usually quite different.

Multiple neuritis is rare in childhood, but occasionally when occurring in an acute form difficulties in diagnosis will arise. Seldom, if ever, will the onset be so acute or sudden; the fever is of longer duration; there are marked sensory symptoms, as extreme tenderness of the nerve-trunks and muscles, and passive movements greatly increase the suffering. The distribution of the paralysis is also helpful, and the history of a recent throat-affection will clear up the difficulty in *diphtheritic neuritis*.

Cerebral palsy is differentiated by its distribution, by the absence of the reaction of degeneration and of marked wasting, and of loss of reflex action.

Acute transverse myelitis is extremely rare in early childhood; myelitis occurring in very early life is almost certain to be limited to the anterior horns. Hence there is no difficulty in the diagnosis, except when the cornua on both sides are involved and the mischief extends into the white columns. The presence of involvement of the sphincters, sensory and trophic disturbances, with spasms, even if wasting be marked, will demonstrate that the lesion is not that of poliomyelitis. Established cases of infantile paralysis can hardly be mistaken for any other disease; the reaction of

degeneration, with the wasting rapidly following a loss of power, which is never progressive though rapid or abrupt, and the absence of reflex action, without evidence of any loss of sensation, form a clinical picture which is unmistakable.

The history of *Progressive muscular atrophy* of the spinal type is always distinctive, showing a slow and insidious onset, with progressive advance, though the site of the lesion is chiefly confined to the anterior cornua, as implied in its synonym of chronic poliomyelitis. The congenital palsies of muscular origin are also of such very slow onset as to prevent an error in diagnosis. (See under 'Pseudo-hypertrophic Muscular Paralysis,' p. 1060.)

Prognosis.—As already stated, there is little danger to life save in the severe acute cases at their onset, and this danger may usually be regarded as having passed by after a period of forty-eight hours or less from the onset, as after the expiration of this time it is rare for the paralysis to go on increasing or extending.

The prognosis regarding the probable amount of permanent damage is of great importance. Usually it can be given with an approximation to accuracy in many cases by a careful electrical examination made about ten days after the onset. If by this time the loss to faradism is complete and the wasting has commenced, little return of power or but a trifling restoration of function need be expected in the affected muscle-groups. A loss of irritability to faradism which only shows itself later than this will probably be followed by very considerable improvement, and in cases where after two or three weeks the reaction of degeneration is still absent in most of the muscles in the affected limb a hopeful prognosis may be safely given; but such cases are rare, and it is a wise rule always to prepare the child's friends for some permanent weakness and shortening of the affected limb. Trevelyan states that if there is no reaction to faradism in two months, permanent loss of power, more or less complete, is to be feared. Very seldom, if ever, does absolute and complete recovery occur in all the muscles. Any marked degree of recovery should be obviously started within twelve or sixteen weeks from the onset, if it is to take place at all.

Treatment.—In the early stage of the disease nothing can be attempted, save that repose as nearly as possible amounting to absolute rest must be rigidly enjoined. If seen during the high-temperature period of the onset, salicylates or, better still, small doses of antipyrin may be administered. No reliance need be placed in dosing with belladonna and ergotin with the view of diminishing the inflammatory action in the horns of the cord; probably strychnine would be positively injurious, and a like objection may be made to the spinal ice-bag. Pain may, however, be

relieved by warm poultices, compresses, or mild counter-irritation.

Rest is still essential for two or three weeks or more after the paralytic symptoms have become evident, and it will be wise to postpone the administration of strychnine for a longer period—say for five or six or eight weeks.

Electricity, massage, and gymnastic exercises are alone to be depended upon to prevent the muscular degeneration progressing; these agents, however, exercise no power whatever over the damaged or ruined nerve-cells. The use of electricity should be confined to the galvanic current, which must be very weak at first. One terminal of the battery, with its sponge well wetted by warm saturated saline solution, should be placed over the nerve entering the upper part of the muscle, and the other used to stroke gently the mass of the muscular fibres, breaking the contact frequently so as to produce mild contraction. In this way the nutrition of the muscle may be kept up, with the hope that when regeneration of partially damaged nerve-cells commences the muscular fibres may be placed in a position to respond to their influence. The negative terminal is best used as the movable one, and as a rule the applications should not be commenced for at least four weeks after the onset. At a late stage only is faradism justifiable, by which time massage and gymnastic exercises are indicated; faradism may be employed at an earlier stage, however, in muscles which still respond to it. The use of these measures often tend to prevent contractures and permanent deformities when careful attention is paid to the influence of posture at the same time. Even after the establishment of contractures much may be done by judiciously planned surgical measures, as the adjustment of mechanical supports, alone or in conjunction with the cutting of tendons.

PARALYSIS, LANDRY'S. (See p. 1165.)

PARALYSIS, PERIPHERAL, OF CEREBRAL NERVES

Under 'Neuritis' are described the results which follow trauma, pressure, or acute inflammation of nerve-trunks. These are always associated with more or less paralysis when motor nerves are involved, though the obliteration of function in the sensory nerves and nerves of special sense is often also spoken of as a paralysis. In the present and succeeding articles a brief reference will be made, for convenience, to the paralysis of the different nerves arising from the brain and spine, the cerebral nerves being first noticed.

The *olfactory nerve* may entirely fail in its function, so that complete anosmia results, as described under 'Olfactory-Nerve Disease,' p. 1137.

Optic-nerve disease is discussed under its own heading, where affections of the disc, retina, chiasma, tract, and nuclei are described (see pp. 1141-1147).

THIRD NERVE.—This nerve is called the oculo-motorius ; with the fourth, sixth, and sympathetic it supplies the external and the internal muscles of the eyeball.

Symptoms.—When this nerve is paralysed all the muscles of the eyeball suffer, save the external rectus and the superior oblique. The ciliary muscle and the sphincter of the iris suffer likewise along with the levator palpebræ. The pupil cannot contract, loss of accommodation, ptosis, and divergent squint occur ; the eyeball can only be turned outwards and slightly downwards and inwards. When the patient attempts to raise the weakened levator the frontalis, with which it normally acts, is brought strongly into action, corrugating the brow on the affected side ; there is double vision.

Often the paralysis is not complete, or only affects certain parts of the nerve, so that the internal muscles may escape whilst the external ones suffer ; when the loss of power in the external muscles is not complete, the levator may entirely escape. When the internal muscles are involved, *cycloplegia*, or loss of accommodation for near vision, occurs, and *iridoplegia of the accommodative type* results, in which the pupil does not contract in looking at near objects. *Reflex iridoplegia*, or the failure of the pupil to contract to light, may be also present.

The paralysis may be caused by an inflammation of the nerve, as in diphtheritic and tabetic neuritis, or it may be produced by a gummatous growth, aneurism, sinus-disease, meningitis, or trauma from a blow on the temple. In the orbit the nerve may be paralysed by a rheumatic neuritis, by a hæmorrhage into its sheath, by a cellulitis or tumour, or by syphilitic disease. In all these cases the paralysis is usually unilateral, but when the pressure is produced by a growth between the cerebral peduncles or by an aneurism of the posterior cerebral both nerves may be involved, though the paralysis is nevertheless peripheral. When the nerve is involved in a unilateral disease of the crus there will be hemiplegia on one side and paralysis of the ocular muscles of the opposite eye ; this '*alternate*' hemiplegia is owing to the fact that the motor tract decussates below the origin of the nerve ; and if the lesion (tumour, softening, sclerosis, or hæmorrhage) catches the cerebral fibres going to the nucleus of the ninth nerve there will be also unilateral paralysis and wasting of the tongue.

Nuclear disease nearly always also involves the origin of the fourth and sixth nerves, so that the symptoms are not only usually bilateral, but there is also a general total ophthalmoplegia, the causes being commonly degenerative in the slow cases, inflammatory in the acute, and sudden in the hæmorrhagic or embolic ones. Peripheral ophthalmoplegia affecting both eyes is not very rare in diphtheritic and alcoholic neuritis, and may be recognised by the involvement of other nerves, especially those of the limbs. (See under 'Ophthalmoplegia,' p. 1138, where nuclear disease is described.)

When the palsy is due to syphilis it is liable to come and go, sometimes involving the third nerve on one side, which clears up after a time, the opposite nerve becoming involved; this *relapsing* form is almost characteristic of a specific infection, and is liable to end in a persistent affection of the nerve first affected; not rarely the only sign in these cases is ptosis. A similar condition is sometimes met with in tabes in which the paralysis is also often transient; seldom or never is it the result of a true peripheral degeneration, being nearly always due to neuritis.

Periodical or recurring paralysis of the third nerve has often been observed, alone or, in rare cases, associated with palsy of the sixth. The attack is usually accompanied or preceded by severe pain at the back of the orbit or in one side of the head, with vomiting. Generally all the branches of the nerve are involved, but the paralysis is not usually total, and after a few weeks it passes off completely. The attacks may be separated by intervals of weeks, months, or a few years. They are more frequent in women, and may commence in childhood and last throughout middle-life. The pathological condition underlying these attacks is unknown, but it is conjectured to be of the nature of migraine.

FOURTH NERVE.—This, the trochlear nerve, only supplies the superior oblique muscle. It arises from the valve of Vieussens in the floor of the fourth ventricle; its nucleus is in immediate connection with the nucleus of the third, and its fibres decussate between its deep and superficial origins. It winds around the outer side of the crus to reach the front of the pons, where it may be involved in a basilar meningitis, tumour, or aneurism; but seldom is it involved alone. The paralysis caused by involvement of this nerve is most difficult of detection; the loss of power shows itself in a diminution of the downward and inward movement of the eyeball, as seen by the failure of the globe to follow the descent of an object below the horizontal line. There is slight convergent strabismus below the horizontal plane which causes a most confusing and annoying double vision *when the patient is looking downwards*, as in walking or reading; the head is inclined towards the sound side and forwards.

FIFTH NERVE.—The trigeminal nuclei and deep roots cover a very extensive area in the brain, reaching from the corpora quadrigemina to the upper part of the spinal cord. The nerve arises by two roots from the side of the pons or inner surface of the middle peduncle of the cerebellum; the smaller anterior root is motor, the large root lying alongside it is purely sensory. The motor-root fibres end in a group of large nerve-cells near to the outer angle of the fourth ventricle. The sensory root is regarded as arising in the Gasserian ganglion, from which the fibres proceed to the pons, some passing inwards towards the opposite side under the floor of the fourth ventricle, some passing upwards as far as the corpora quadrigemina, but the majority of them descend through the pons and medulla to form connections with the ramifications from the cells in the posterior horns of the cord, thus really constituting the posterior spinal roots.

The trigeminal thus resembles a spinal nerve in having both sensory and motor functions. The sensory fibres, as already stated, are in connection with the Gasserian ganglion (lying upon the depression on the petrous bone), in which they form a plexiform web. From the convex border of the ganglion three large branches issue: the first—the ophthalmic—goes to the orbit; the second—the superior maxillary nerve—passing through the foramen rotundum, reaches the face; both these nerves are purely sensory. The third branch—the large inferior maxillary nerve—after issuing from the ganglion, is joined by the small motor root of the fifth nerve, and, thus becoming a mixed nerve, divides into two chief branches, the upper of which, being mainly motor, supplies the muscles of mastication. The lower branch—mainly sensory—gives off the lingual or gustatory nerve for the tongue, the inferior dental for the teeth, and the auriculo-temporal branch for the side of the head.

Etiology.—Functional disease of the nerve, causing neuralgia, is described upon page 1094. Primary disease of the nerve, causing paralysis, very rarely occurs, and inflammation seldom originates in its trunk or branches; but secondary involvement of the nerve is by no means rare from injuries and diseases at the base of the brain, from meningitis, caries of the temporal or sphenoidal bones, syphilitic disease, and tumours, and sometimes from hæmorrhagic or degenerative changes in the pons and medulla. The branches may be also secondarily involved in their outward course; thus the ophthalmic may be pressed upon by tumours about the orbit and by aneurisms of the internal carotid and by the products of sinus-diseases or cellulitis. The second division and the inferior maxillary trunk are liable to be involved by growths springing from the bones forming the middle fossa of the base of the skull, by trauma, and by malignant and

other tumours occupying the spheno-maxillary fossa or springing from the parotid region. Diseases of the spinal cord, as in tabes, may also involve the sensory part of the nerve, but rarely the motor portion.

Symptoms.—In total paralysis of the trunk of the trigeminal the *sensory* symptoms are anæsthesia of one half of the face and of the head, with loss of sensation in the mucous membrane covering the front of the eye, the interior of the nose, and the mouth, with the tongue, palate, and gums. The anæsthesia is generally preceded by smart neuralgic pains and tinglings. The tears and nasal mucus are diminished so that more or less anosmia results. The corneal and lid reflexes are absent, so that the unprotected cornea is liable to become inflamed and ulcerated; though when this form of keratitis occurs it is maintained by Turner and others to be of *neuritic* origin: it certainly cannot be regarded as a trophic change, since it very rarely follows extirpation of the Gasserian ganglion.

Pungent vapours fail to induce sneezing, and tickling of the palate does not cause efforts at retching. The facial muscles, though scarcely, if at all, weakened in their action in any way, are more or less insensible.

The sense of taste may be entirely absent; more frequently it is normal over the posterior third of the tongue. Herpes, more especially in the area of the ophthalmic branch, may be severe and accompanied by intense and lasting pain, and other trophic changes may involve the inside of the mouth, teeth, or nostril. The cheek is liable to be bitten in mastication and to heal slowly, the teeth may loosen, and one side of the tongue only may be covered with fur.

The *motor* signs occur when the motor root or the inferior maxillary trunk is affected. The muscles of mastication are paralysed; the temporal and masseter on the affected side do not contract in the act of biting, as demonstrated by placing the finger-tips over them when the mouth is firmly closed. The lower jaw cannot be moved towards the sound side by the external pterygoid, and when the mouth is wide open the jaw hangs towards the affected side.

Atrophy of the muscles may ultimately follow, and the electrical reaction be of the degeneration type in peripheral cases; sometimes facial hemiatrophy has been observed to supervene later on, the bones and subcutaneous tissues suffering always more than the muscles.

The paralysis of the tensor tympani and palati, of the mylohyoid and digastric, causes no disturbance of motility or other symptoms.

The opposite condition to paralysis—viz. spasm or trismus—may be occasionally found to precede the paralysis when a nuclear lesion of a distinctly *irritative* nature is present; it

may be the result of some stimulation of a sensory peripheral branch of the nerve which by reflex action throws the masticatory muscles into spasmodic contraction. Such a condition is generally only seen in cases where the injury to the nerve is very partial, as in alveolar disease.

Diagnosis.—This is easy, as a rule, but *hysterical paralysis* may be confused with it when there is anæsthesia of one side of the face and head, but a careful examination will always reveal anæsthesia in other parts in hysteria. When the lesion is mainly irritative, the disease may be mistaken for *neuralgia*, till the advent of anæsthesia clears it up; but even before this there may be found absence of taste, which is almost equally conclusive. When the paralysis of sensation and motion is complete within the entire area of distribution the lesion is probably at the base of the brain or involves the Gasserian ganglion. When the mischief is located in the middle fossa of the skull the ophthalmic division is usually alone affected. Lesions (hæmorrhage, tumour, &c.) about the side of the pons usually catch the sixth or third nerves at the same time that all the divisions of the fifth are involved. Lesions inside the pons cause, in addition to complete involvement of the trigeminal, hemiplegia on the opposite side and inability to move both eyeballs towards the side of the pontine lesion. When the chorda tympani is alone involved there is loss of taste on one side in the anterior two-thirds of the tongue, without anæsthesia, but when the lingual nerve is involved after its junction with this nerve there is loss of taste, whilst a lesion of the third division above the junction may be followed by no diminution of this sense with anæsthesia. Loss of taste occurs also in facial paralysis when the nerve is involved between the origin of the chorda tympani and the geniculate ganglion—a fact worth remembering, since the facial and even the auditory are sometimes also involved in paralysis of the trigeminal.

The prognosis and treatment depend upon the nature of the lesion involving the nerve. The rare cases of rheumatic origin may recover, as in facial paralysis; pain must be relieved by the measures suitable for neuralgia. Faradisation of the skin may be tried for the restoration of function when there is anæsthesia without pain. Antisyphilitic remedies should always have a trial, and the eye should be carefully guarded against injury by foreign bodies and direct exposure to cold.

SIXTH NERVE.—The abducens nerve only supplies one muscle—the external rectus. Its nucleus is situated beneath the eminentia teres in close connection with the facial nucleus. Its surface-attachment is between the pyramidal body and the pons, from which point it passes forwards in the floor of the cavernous sinus by the side of the carotid artery, to enter

the orbit through the sphenoidal fissure. In its long and exposed course it is more frequently involved than any other nerve in the diseased processes already referred to in the description of the other ocular palsies.

Symptoms.—These are seen in the limitation of the outward movement of the eyeball, the strabismus being convergent and liable to increase from secondary contracture. Diplopia occurs in the corresponding outer half of the field of the affected eye; the two parallel images are upon the same level when the object looked at is held horizontally in front of the eye, and as it is moved outwards the images separate. The head is turned or inclined towards the side on which the paralysis has occurred, in order to avoid the homonymous or simple diplopia; the secondary deviation is by the opposite internal rectus.

In nuclear lesions the seventh nerve may be also affected upon the same side. The *internal* rectus of the opposite side (supplied by the third nerve) is paralysed in disease of the sixth nucleus in the pons, because the nucleus of the sixth nerve acts upon the opposite third nucleus and internal rectus through the posterior horizontal fibres connecting both nuclei together. Hence a pontine lesion on one side produces a loss of movement of both eyes towards the side upon which the lesion is situated, or, in other words, conjugate deviation of both eyes occurs towards the sound side of the brain. In ordinary hemiplegia the reverse condition occurs, both eyes looking towards the side upon which the lesion is situated. (See also under 'Ophthalmoplegia,' p. 1138.)

SEVENTH NERVE.—Paralysis of this nerve is already described under 'Facial Paralysis,' upon p. 414.

EIGHTH NERVE.—The auditory nerve, sometimes called the eighth nerve or the portio mollis of the seventh pair, is exclusively distributed to the internal ear. Its two divisions possess different functions, the vestibular branch supplying the vestibule of the inner ear by which the body-equilibrium is regulated, and the cochlear division being responsible for the function of hearing. Paralysis or loss of function in this nerve causes deafness and other symptoms. The subject is dealt with under 'Auditory-Nerve Disease,' upon page 97, and also under 'Vertigo.'

NINTH NERVE.—The older nomenclature, by which the eighth pair of nerves included the glosso-pharyngeal, pneumogastric, and spinal accessory, is here departed from.

The Glosso-pharyngeal, or ninth nerve, is intimately associated with the nuclei of the pneumogastric and spinal accessory, situated deeply in the floor of the fourth ventricle. The sensory fibres of the nerve arise in the petrosal and other ganglia outside the medulla, forming the descending sensory vago-glosso-pharyngeal root, which is known as the solitary

fasciculus; whilst a small collection of large cells found in the upper part of this is supposed to constitute its motor nucleus. The surface-attachment of the nerve is at the restiform body of the medulla, where its series of small superficial roots are in line with those of the facial and auditory above and with those of the pneumogastric below.

The nerve is distributed to the posterior third of the tongue and the whole of the sensory pharynx, and also supplies most of the pharyngeal muscles. Its functions are not accurately known, as the nerve is never found to be paralysed alone.

Symptoms.—Loss of taste at the back of the tongue has not yet been demonstrated to be caused by disease of this nerve; taste has been found to be normal when it has been extensively diseased, and loss of taste over the entire tongue has been observed, as already described, when the disease was confined to the fifth nerve. Hence it has been held that the glosso-pharyngeal fibres supplying the back part of the tongue leave the nerve and join the fifth through the otic ganglion or the tympanic plexus, or they come from the fifth nerve. The nucleus of the nerve is seen to be involved with the other nuclei in bulbar paralysis, and the peripheral part may be affected by syphilitic disease, aneurisms, thrombotic changes, tumours, bone-disease, &c. There will be loss of sensation in the upper part of the pharynx, and of reflex excitability, with dysphagia, or inability to swallow, from motor paralysis, and probably there will be loss of taste in the posterior third of the tongue.

TENTH NERVE.—The pneumogastric has its surface-attachment at the restiform body on the side of the medulla, where its numerous superficial roots issue below and in line with those of the glosso-pharyngeal. From the surface the fibres can be traced inwards to the nucleus lying deeply in the floor of the fourth ventricle, where they meet the nucleus of the opposite side in the middle line at the calamus scriptorius. It has the longest course and most extensive distribution of all the cranial nerves, and supplies the pharynx, œsophagus, larynx, lungs, heart, spleen, stomach, and intestines. The nerve possesses both motor and sensory fibres. The latter are supposed to originate in conjunction with the glosso-pharyngeal in the solitary fasciculus, whilst the motor fibres are conjectured to arise in the ambiguous nucleus.

Its pharyngeal branch joins with the glosso-pharyngeal and sympathetic in forming the plexus which supplies the muscles and the mucous membrane of the pharynx. The laryngeal branches, superior and inferior, are of great importance, since they supply the laryngeal muscles (they are supposed to be mainly derived from the spinal accessory). Paralysis of the pharyngeal branch is included in the description of the symptoms of bulbar paralysis on page 1175, and the

palsy which follows interference with the laryngeal branches is described under 'Laryngeal Paralysis.'

The afferent fibres, which exercise an accelerating and inhibitory influence over the respiration by reflexly stimulating the respiratory centre, control the respiratory movements. Section of the nerve-trunk causes the breathing movements to become slower and deeper, owing to the more powerful influence of the accelerating fibres being withdrawn; and a similar result is obtained by stimulating the superior laryngeal which contains the chief inhibitory fibres going up to the respiratory centre.

Section of the nerve produces marked acceleration of the heart-movements, since its cardiac function is mainly inhibitory, but it also contains accelerating fibres. The fibres going to the lung are both sensory and motor, the latter supplying the muscular tissue of the bronchi and the former the mucous membrane.

The stomach is supplied by secretory, sensory, vaso-motor, and motor fibres, and the intestines also receive part of their motor supply from it. The influence of its afferent fibres over the vaso-motor centre is important; upon stimulation of the nerve dilatation of the arterioles throughout the body occurs, resulting in fall of the blood-pressure.

The nucleus of the nerve may be affected by diseases occurring in the medulla; but the other bulbar nuclei are then certain to suffer also, and the symptoms are described under 'Paralysis, Bulbar,' 'Ataxia,' 'Disseminated Sclerosis,' 'Cerebral Hæmorrhage,' &c.

In its intracranial course the nerve may be involved by the products of meningeal inflammation, tumours, disease of bones, sclerosis or aneurismal dilatations of the vertebral and inferior cerebellar arteries, and by syphilitic disease or sinus-thrombosis, in which cases also other cerebral nerve-trunks are very liable to suffer at the same time. Peripherally the nerve may suffer from diseases or injuries, accidental or surgical, in the neck, from pressure in the mediastinum by glands, and aneurisms, or by dilatation of the auricle in mitral stenosis, pericardial effusion, &c.

Neuritis of the pneumogastric is rarely primary, but it suffers often in both diphtheritic and alcoholic neuritis, and may be involved in the multiple neuritis caused by any mineral or organic poison.

Symptoms.—These depend largely upon the site of the lesion in the course of the nerve, as also upon its nature, *irritative* lesions causing spasm, whilst those which produce entire loss of conducting power cause total paralysis. Thus an irritative lesion will cause laryngismus stridulus or laryngeal spasm when the nerves going to this region are over-stimulated, whilst a similar affection, by acting upon the gastric supply,

may produce vomiting and pain, in marked contrast to the various paralytic phenomena described under 'Laryngeal Paralysis,' and to the loss of motor power in the stomach, which occurs when the gastric fibres are paralysed. Irritation of the pulmonary branches may result in attacks of asthma, whilst paralysis of these fibres will eventuate in pulmonary congestion, hæmorrhages, and aspiration-pneumonia. Cheyne-Stokes respiration, excessive slowing or great acceleration of the cardiac rhythm, will arise, dependent upon the portion of the fibres stimulated or weakened, the paralysis of the inhibitory fibres showing itself in the loose rein given to the accelerators. Unilateral lesions, however, usually fail to cause marked disturbance of either heart, lungs, or stomach, if the laryngeal affections are excepted. Spasm of the œsophagus arises when the pharyngeal branches are irritated, as in hysteria; but paralysis of these branches by organic lesions will cause difficulty of swallowing through weakness of the muscles, as seen in the typical condition of bulbar paralysis in which the food lodges in the pharynx, or may gain access to the larynx, causing spasm, aspiration-pneumonia, &c.

When only one pharyngeal branch is involved the symptoms are slight or not noticeable; they are seen in unilateral paralysis of the faucial muscles and palate. One vagus-trunk only being paralysed, the larynx suffers with the pharyngeal muscles and palate; the voice is low-pitched, hoarse, or nasal, and the paralysed vocal cord remains motionless in the cadaveric position; but anæsthesia of the pharynx is seldom appreciable.

Functional affections of the vagus are seen in hysteria, under which heading their symptoms are described. The laryngeal aphonia and paralysis resulting from defective phonic adduction in this neurosis is detailed under 'Laryngeal Paralysis,' and functional disturbances affecting the cardiac rhythm are referred to under the respective headings in the description of functional affections of the heart.

The prognosis and treatment depend entirely upon the nature of the organic lesion or functional disturbance affecting the nerve, its nucleus or branches, and they are referred to under their respective headings.

ELEVENTH NERVE.—The spinal accessory nerve has its surface-attachment—(1) by a series of superficial roots arising below those of the pneumogastric upon the side of the medulla; these—the *accessory* or *internal* fibres—join the pneumogastric trunk beyond its second ganglion. Traced inwards, they reach a nucleus of large cells near the middle line outside the eminentia teres, where they are in intimate connection with the nucleus of the vagus and glosso-pharyngeal. These fibres, forming the upper series of the superficial roots, when they join the vagus go to innervate the

laryngeal muscles, and their involvement produces part of the clinical picture of vagus paralysis or irritation. (2) The spinal accessory possesses a series of lower superficial roots arising from the lateral column of the cord as low down as the sixth or seventh cervical nerves. These—the *external* part of the nerve—when traced inwards are found to arise from a group of cells in the anterior cornua of the cord. As the fibres of the external part of the nerve pass outwards through the jugular foramen (after having first entered the cranium), they communicate with the accessory portion, and are finally distributed as spinal nerves to the sterno-mastoid and trapezius muscles.

The above is the usually accepted view of the distribution of the accessory portion of the nerve, but Grabower maintains that the laryngeal muscles are innervated through the lower root-bundles of the vagus arising from the ambiguous nucleus. Hence Hall and Grabower regard the eleventh nerve as *entirely a spinal nerve*, the accessory branch being a portion of the vagus from its very origin, as accepted by Oppenheim.

The spinal part of the nerve may be damaged in cervical caries, pachymeningitis, tumours, syphilitic disease, degenerative changes in the upper part of the cord, as in tabes, muscular atrophy, &c. Rarely does the nerve become affected in neuritis of the peripheral type, and it does not inflame primarily.

Symptoms.—When the lesion is of an irritative nature, spasm of the affected muscles produces torticollis. Paralysis of the sterno-mastoid and, to a lesser extent, of the trapezius follows disease of this spinal nerve. The sterno-mastoid paralysis produces no symptoms whilst the head is at rest, but the loss of power is followed by marked wasting, and the head and chin cannot be rotated to the opposite side; any attempt at the execution of this movement displays the inertia of the affected muscle, which, moreover, does not work in forced inspiration. When the unopposed sterno-mastoid becomes the seat of contracture the head may be markedly deviated. The paralysis of the trapezius is usually confined to the upper portion of the muscle, which causes a characteristic deformity by producing a concave contour of the outer side of the neck, and the weakened or paralysed muscle does not act in forced inspiration; the arm cannot be elevated as well as upon the sound side when the second portion of the muscle is involved and the shoulder is depressed. The angel-winged deformity of the scapula is, however, not observed, as the middle portion of the trapezius is never completely paralysed.

When the paralysis of the sterno-mastoid is bilateral the head tends to fall backwards, whilst if both trapezii are

affected the chin may fall forwards and rest upon the sternum. The reaction of degeneration is usually well marked, except when the paralysis is but partial. In lesions affecting the nerve before it finally emerges from the cranium the hypoglossal is often involved ; and if the spinal portion is affected along with the accessory there may be laryngeal symptoms as well.

The prognosis and treatment, as in other forms of peripheral palsies, depend entirely upon the nature of the lesion ; the outlook is generally fair in syphilitic cases. Electrical stimulation is useful in keeping up the tone of the paralysed muscles whilst the antisyphilitic or other treatment is being vigorously pursued.

TWELFTH NERVE.—The hypoglossal nerve is the motor nerve for the tongue ; it also supplies most of the muscles attached to the hyoid bone. The superficial origin of the nerve is by a series of slender roots from the sulcus between the olivary body and the anterior pyramid of the medulla. Traced inwards, they are seen to reach the nucleus, which is close to the raphé lying in front of the central canal as low as the decussation of the pyramids ; the nucleus at its upper part lies in the middle line in the floor of the fourth ventricle. The nerve in the cranium and in the upper part of the neck lies in close relation to the pneumogastric and spinal accessory, joining the petrous ganglion of the former nerve and receiving or giving fibres to the loop between the first two cervical nerves.

Paralysis of this nerve is more frequently central than peripheral, and hence it is commonly bilateral, forming part of the clinical and pathological picture in bulbar paralysis, since the nuclei of both sides lie together. *Supranuclear* disease commonly affects the nerve, so that it is very frequently involved in ordinary hemiplegia, the fibres being injured in the motor tract at any level between the cortical centre and the nucleus of the nerve ; but in these cases it is worth remembering that, though the tongue-muscles are paralysed on one side, they do not waste, since the nerve is not severed from its relations with its medullary trophic centre.

In the medulla the *infranuclear* fibres may suffer from tumour or pressure, and in one rare case seen by the writer this was unilateral. Peripheral palsy of the nerve may be caused by bone-disease, tumour-pressure, or by mischief in the posterior fossa of the skull, syphilitic, aneurismal, hæmorrhagic, or meningeal. Rarely is it induced by tumours, abscesses, or trauma in the upper part of the neck, but occasionally it is involved secondarily in a multiple neuritis. When paralysis of one nerve occurs the tongue is weakened and wasted on one side ; with the mouth opened widely, the organ is seen in its usual position or with its tip very slightly

inclined to the sound side. When the tongue is protruded it deviates to the paralysed side owing to wasting of the genioglossus, but the point may be slightly deviated or twisted round to the sound side. Fibrillary twitchings may be observed in the wasted half of the organ, which is flaccid and wrinkled. When the paralysis is bilateral the tongue cannot be moved to either side or protruded, and articulation is markedly indistinct; but in unilateral palsy this function is slightly, if at all, disturbed, and neither deglutition nor mastication is interfered with. In neither form is sensation or taste impaired, the symptoms being entirely motor.

The site of the lesion is located by the above symptoms, nuclear disease being commonly bilateral and associated with paresis of the lips and pharyngeal muscles, followed by wasting. When the lesion involves the fibres in the medulla below the nucleus, the motor tract of the opposite side is usually involved, so that the tongue-paralysis and wasting are on the side opposite to the hemiplegia; but in the supra-nuclear disease the tongue is weakened and not atrophied upon the hemiplegic side, and when protruded is deviated to this side. A unilateral lesion involving the superficial roots arising from the surface of the medulla will probably also catch the pneumogastric and spinal accessory roots, so that, in addition to the hemiatrophia linguæ, there will be unilateral paralysis of the velum palati and of the larynx; such a condition is very likely to be of syphilitic origin. The prognosis and treatment are identical with those of the other cerebral nerve-palsies, and will depend entirely upon the nature of the lesion which has arrested the function of the nerve.

PARALYSIS, PERIPHERAL, OF SPINAL NERVES

Under 'Neuritis,' upon page 1114, the multiple form of inflammation of the peripheries of the nerves is described. It is usually of toxic origin, and, since it ends in motor paralysis, it is often known by such names as alcoholic, arsenical, mercurial, diphtheritic, or influenzal paralysis; beri-beri is another form of the same paralytic phenomenon.

The spinal nerves spring from the cord each by two roots—a small anterior and a large posterior, which join into one trunk in the intervertebral foramen. The posterior root, which is sensory, has a ganglion upon it as it lies in the foramen, but the anterior is devoid of any ganglionic enlargement, and joins the posterior root on the distal side of the ganglion.

The nervous system may be regarded as made up of a countless number of neurons or units. Each neuron consists

of a nerve-cell or ganglion-cell, from the extremity of which are given off small branches or arborisations, known as *dendrites*, which convey impulses to the cell-body from other dendrites. From what, for convenience, may be termed the distal pole of the nerve-cell passes the *axon* or axis-cylinder, which ultimately becomes a nerve-fibre and gives off small collateral twigs at right-angles. The axon conveys impulses *from* the cell-body, which is regarded as the originator or reservoir of nerve-force, and it finally terminates in numerous fine brush-like arborisations.

The *entire motor path* may be regarded as originating in the large motor-nerve cells in the motor area of the cerebral cortex; from these, through their axons, which decussate in the medulla, cellulifugal impulses pass downwards to the ganglion-cells of the nuclei of the cranial nerves, and also to the elements of the spinal cord which correspond to these—viz. to the large ganglion-cells in the anterior horns. The axon divides into its end-brush or terminal arborisations, which freely *intermingle* with the dendrites given off by the ganglion-cells of the cord; but they are supposed to end free and only to come into contact, without structurally uniting. Each of the ganglion-cells of the cord and of the brain-nuclei gives off by its distal pole an axon or axis-cylinder, which, passing outwards in the cords of the cranial motor nerves and in the spinal nerves through their anterior roots, finally end in the terminal muscle-plates.

The spinal nerves are therefore to be regarded as the lower segment of the motor path, containing the axons derived from the ganglion-cells of the anterior horns. The impulses originating in the cerebral cortex pass first through the central neurons of the upper segment of the motor path, which terminate in their brush-ends before reaching the cells in the anterior horns, the impulses finally reaching the muscles by passing downwards through the peripheral neurons of the lower segment.

Disease of the cell-body will cause degeneration of its dendrites and of the axon proceeding from it, as it exercises a trophic function on both sets of processes; and it will itself also suffer if cut off from its dendrites and axons. The sensory path passes through the neurons in the ganglions of the posterior roots. (See under 'Paralysis, Spastic,' where the symptoms of disease of the different portions of the motor tract are contrasted.)

In studying paralysis of spinal origin it must be remembered that the nerves in the cervical, lumbar, and sacral regions are so interlaced in plexuses that the nerve going to a muscle or group of muscles is represented in more than one segment of the cord. The term 'spinal segment' is usually applied to that portion of the cord with which each pair of spinal nerves

is connected, independent of the relation of the segment to the contiguous vertebral bodies. Hence disease of a portion of a plexus or of a nerve-root is much more likely to affect groups of muscles than a single muscle, which latter may be the case when the paralysis is caused by pressure, injury, or disease of a nerve-trunk below the plexus.

As already stated, paralysis originating in the spine usually involves both sides of the body, and upon page 1164 the differences between spinal and peripheral paralysis are detailed and their relations to the reaction of degeneration commented upon. In peripheral paralysis, the spinal nerves being mixed cords containing both motor and sensory fibres, loss of sensation accompanies the loss of motor power. This is usually a marked feature in the paralysis of multiple neuritis, where the loss of power is accompanied by anæsthesia and sometimes by hyperæsthesia or other sensory disturbances, in contrast to the infantile type of spinal palsy, where sensation is intact. Muscular dystrophies are characterised by absence of the reaction of degeneration, since the disease is primarily of muscular origin, and there is apparent hypertrophy. Hysterical paralysis may closely simulate any form of loss of motor power, cerebral, spinal, or peripheral. The paralysis is simple or flaccid ; there is no reaction of degeneration or marked wasting, but the distribution of the anæsthesia and contractures, together with the psychic disturbances, are characteristic. Monoplegia of the upper limb is seldom spinal ; it is generally cortical, or the result of neuritis or of hysteria. Monoplegia of the leg, upon the other hand, is seldom cerebral, being due either to neuritis, anterior poliomyelitis, or, more rarely, to unilateral myelitis.

The term *ischæmic paralysis* is usually applied to the form of palsy which affects a limb after tight bandaging or the application of splints or surgical appliances causing pressure upon the nerves and muscles, interfering with their blood-supply.

The more important varieties of peripheral paralysis are the following :

PHRENIC NERVE.—Paralysis of this nerve, which arises from the fourth cervical and usually receives a fasciculus from the fifth, may be due to disease of the spinal cord and its membranes in the cervical region, or to injury or disease of the roots or of the trunk of the nerve itself. In the former case other spinal nerves are certain to be implicated at the same time, and the paralysis will be bilateral. When the paralysis is part of the result of a multiple peripheral neuritis both nerves may be also affected at the same time ; but a single phrenic may be paralysed by a neuritis of the rheumatic type, by the pressure of tumours in the neck or thorax, or by deep wounds in the cervical region.

Symptoms.—The nerve supplies the diaphragm, and when paralysed upon both sides this muscle loses all power, the breathing being then carried out by the action of the intercostals and accessory inspiratory muscles. The upper part of the abdomen, instead of being protruded in inspiration, is retracted, and it can be noticed by palpation that the abdominal viscera do not descend; inspection shows that the thoracic expansion is greatly increased, especially in the lower zone. When the patient lies at rest the effects of the loss of power may entirely escape observation, unless he be suffering from bronchial catarrh. During movements of the body there may be dyspnoea or very rapid breathing, and any attempt at sustained phonation shows weakness in the voice. Paralysis of one half of the diaphragm may give rise to no symptoms, unless in deep inspiration, when it may be noticed that the affected side does not descend. The tendency is ultimately towards blood-stasis in the lungs and the accumulation of mucus in the lower bronchial tubes—a condition which in alcoholic or diphtheritic neuritis may not attract attention till within a short period before the supervention of acute bronchitis or death from suffocation. The loss of coughing-power and the noiseless type of the cough should arouse suspicion of the bilateral phrenic paralysis in such cases.

The prognosis and treatment depend upon the primary cause; the out-look in multiple neuritis is always grave. Electro-therapy is of little use, but combined with strychnine hypodermically the fatal termination may be postponed for a time, or it may be sometimes entirely prevented by allowing time for the neuritis or other morbid process to resolve. The writer has witnessed the successful results of such treatment in diphtheritic paralysis. Counter-irritation over the nerve in the neck may be tried.

BRACHIAL PLEXUS.—The first dorsal and the anterior branches of the four lower cervical nerves (fifth to the eighth), with a fasciculus from the fourth nerve in the cervical plexus, form the plexus or interlacement of nerves from which the shoulder and arm muscles are supplied. Aneurisms, tumours, blows, or severe contusions, lacerations, dislocations of the shoulder or clavicle, fractures, &c., may cause paralysis of all the muscles supplied by the entire plexus, or of single muscles or groups of muscles. Deep sleep, especially during alcoholism, may produce the same result by causing postural compression. Neuritis may be primary, affecting the entire plexus or its branches; it is then likely to be of the rheumatic type, or it may be the result of an ascending neuritis from disease of any branch. More frequently the cords are involved in a multiple neuritis, in which latter case the distribution of the paralysis is more or less characteristic, as described under

'Neuritis.' Disease of the nerve-roots will also cause paralysis, but as this is most commonly due to vertebral disease and syphilitic cellulitis, the motor palsy is commonly associated with pain, hyperæsthesia, spasm, &c.

The *Posterior Thoracic Nerve*, or external respiratory of Bell, arises from the fifth and sixth nerves and supplies the serratus magnus after a long and exposed course. Injury to the nerve in the neck is commonly produced by the compression of heavy objects carried on the shoulder or by severe muscular exertion, in which cases a traumatic neuritis is set up which usually causes smart neuralgic pains, followed by paralysis of the serratus magnus. Men are obviously much more frequently affected than women, and strong labourers or hammer-men are especially liable to suffer.

Symptoms.—With the shoulder at rest the position of the scapula is little changed, but when the arm is moved forwards the posterior margin of the shoulder-blade recedes from the thoracic wall, leaving a deep groove behind it, and the bone is rotated, its lower angle being raised and brought nearer to the spine. A difficulty is observable in the elevation of the arm above the level of the shoulder, which then has to be raised by the middle portion of the trapezius. The reaction of degeneration may be marked, but there is little, if any, cutaneous insensibility.

The prognosis is always good, but this paralysis is usually most tedious in its progress towards recovery. The treatment is that indicated in ordinary neuritis—rest in the early stages, with electro-therapy and massage at a later stage.

The *Suprascapular Nerve* may suffer from similar causes to those just mentioned, but its isolated paralysis is very rare; not unusually, however, it suffers with the circumflex. Both the supra- and infra-spinatus muscles are paralysed, and the wasting of the latter is usually very obvious; the scapula is rotated as in the last-mentioned form of paralysis, the outward rotation of the humerus is diminished, and many minor movements, as those required in rapid writing, may be interfered with.

The *Circumflex Nerve*, arising from the posterior cord of the plexus, supplies the deltoid and teres minor muscles. It is liable to injury from falls and contusions and from the compression caused by using crutches, and occasionally it suffers in lead-neuritis and in rheumatism.

Symptoms.—Paralysis of the deltoid muscle is the main result of this affection, so that the arm cannot be abducted, and if raised by assistance to the horizontal position it cannot be moved forwards or backwards. When the patient attempts to raise the arm the shoulder is elevated by the trapezius and the scapula rotated, whilst the limb hangs helplessly in the

vertical position. The loss of power in the *teres minor* is of no consequence.

This form of palsy may be entirely motor, often no diminution of the cutaneous sensibility being observable. The shoulder-joint, having lost the support of the muscle, becomes relaxed, but adhesions sometimes form, so that ankylosis of the joint follows, and this, with the wasting which accompanies it, may be readily mistaken for a primary rheumatic affection, which tends to produce identical signs; the history and the absence of the reaction of degeneration will clear up such a case. Deltoid palsy pure and simple is at once differentiated from a rheumatic ankylosis by passive movement, which in the latter case will move the scapula along with the arm, and the reaction of degeneration will be absent; moreover, in these rheumatic cases, even of long-standing, the patient may be able voluntarily to cause contraction of the muscle, which, however, does not effect any movement in the limb.

The *Musculo-cutaneous Nerve*, derived from the cords in the plexus which can be traced to the fifth, sixth, and seventh nerves, is very seldom paralysed alone, except by wounds. The chief symptoms are weakness of the *coraco-brachialis* and *brachialis anticus* and paralysis of the *biceps*, with loss of sensation on the outer half of the forearm. When the arm is supinated, flexion is feeble or impossible when all the three flexors of the elbow are paralysed, but in other positions the *supinator longus* aids the flexion.

The *Musculo-spiral Nerve* may be traced back through the posterior cord of the plexus to the fifth, sixth, seventh, and eighth cervical nerves and forwards to its distribution in the *triceps*, *extensors* of the elbow, wrist, and fingers, and in the two *supinators*, to the skin of the lower part of the arm and the forearm, and through its radial branch to the dorsal aspect of the thumb and next two fingers.

The nerve is the largest offset from the brachial plexus, being the *extensor-nerve* of the arm, though it sends a small branch to the *brachialis anticus*, which muscle is, however, chiefly supplied by the *musculo-cutaneous nerve*. From its position as it winds round the humerus it is very liable to compression in sleep, especially when the patient lies upon the ground and when the weight of the head or trunk rests upon the upper extremity; at the upper part of its course it is liable to be injured by the use of crutches. It is therefore more likely to be paralysed alone than any other nerve of either extremity, trauma being the main etiological factor, crutch-pressure and alcoholic narcosis supplying the largest number of cases. Severe muscular exertion in any sudden extension of the arm, dislocations, fractures, and disease of the humerus, wounds or contusions, also may affect it.

Multiple neuritis seldom involves the nerve alone, but in lead-poisoning, as already described, partial paralysis of this nerve forms an important portion of the clinical picture. Rheumatic neuritis of the musculo-spiral is rare.

Symptoms.—These depend upon the site of the trauma which induces the neuritis from which the paralysis ultimately results. When this is high up, the extensors of the elbow and wrist, the extensor communis digitorum and minimi digiti, abductor longus pollicis, extensors of the thumb, and both supinators suffer.

In pressure on the trunk of the nerve as it lies upon the humerus, after the triceps has been supplied, this muscle escapes; this is the most common type of the paralysis, save the crutch-palsies, when the triceps is then also usually involved. The supinator longus is sometimes unaffected. The hand is flexed fully at the wrist, constituting the characteristic drop-wrist or drop-hand so often seen in lead-poisoning. The fingers and thumb are flexed at their metacarpophalangeal joints and cannot be extended; but if the wrist be extended passively or placed upon a support the other phalanges may be extended by the action of the unparalysed interossei; the fingers may be adducted and abducted, and the wrist-joint lateral movements can also be executed in this position.

When the triceps is paralysed the elbow cannot be extended, unless the limb is placed in such a position that the weight of the forearm and hand, acting by gravity, may cause the movement.

When the paralysis of the extensors is complete, though the flexors are intact, the grip of the hand is weakened, unless the wrist is held in the position of hyper-extension. The paralysis of the supinators is demonstrated by the arm being brought into the position of pronation, unless the elbow is flexed, in which latter case the biceps comes to the rescue or the humerus is rotated outwards by the infraspinatus, thus partially restoring the supinator position. The paralysis of the supinator longus weakens the movement of flexion of the forearm, and if the forearm is placed in the mid-position between pronation and supination forcible flexion does not cause the prominent mass of the muscle to bulge as it does in the sound forearm.

The extent of the anæsthesia varies much. Usually it is only slight and often it is entirely absent, especially in paralysis from simple pressure, as in crutch-palsy; the skin over the dorsal region of the thumb and next two fingers is somewhat numb in most cases. The symptoms of inflammation in the nerve may be severe—viz. pain, tingling, and numbness—before the final loss of power is obvious. In severe cases, where the nerve is involved high up in its course, the entire

cutaneous area of the musculo-spiral supply may be insensible ; but this is rare. The tendon-sheaths or synovial sacs upon the back of the wrist become distended and the carpal bones appear more prominent. The muscles in simple pressure-cases waste but slightly and there is no reaction of degeneration, but in severe injuries there may be extensive wasting and loss of excitability to faradism, and the unopposed pronators may become contracted.

Diagnosis.—*Lead-palsy* is excluded by the rapid onset, unilateral distribution of the paralysis, and by the implication of the supinator longus, which very usually escapes in lead-poisoning.

Prognosis.—Where there is much wasting and the reaction of degeneration is established the recovery will be slow, and probably not complete for a very long period. Mild pressure-cases, as in crutch-palsy, recover rapidly upon withdrawal of the cause. Laceration of the nerve and severe wounds involving its trunk will probably cause permanent paralysis, unless surgical treatment be undertaken at an early stage.

Treatment.—This is to be carried out upon the lines of the treatment suitable for neuritis—absolute rest at first, with massage and electricity at a later stage. Deformities are to be guarded against by supervision of the posture of the paralysed arm and hand. Where the continuity of the nerve-trunk has been severed, suturing of the divided ends of the nerve should be performed ; its distal end may be sutured to the median nerve when its own proximal and distal ends cannot be brought into contact with each other.

The Ulnar Nerve.—This nerve springs from the inner cord of the plexus and derives its fibres from the last cervical and first dorsal roots. It is often involved in company with the median or other nerve, but its isolated paralysis is not uncommon, being caused by dislocations, wounds, and fractures at the elbow-joint. Primary neuritis is rare, but the nerve may suffer in any form of toxic multiple neuritis. Simple pressure-palsy is not uncommon, and may be caused during sleep or in the waking hours by resting the arm against hard substances in such positions as permit of pressure on the nerve.

The ulnar supplies the skin to a very limited extent above the wrist, but the inner and outer sides of the little finger and ring-finger and the contiguous side of the middle digit receive their cutaneous supply from it. It innervates the flexor carpi ulnaris, the inner half of the deep flexor of the fingers, the interossei of the hand, the two internal lumbricals, the short muscles of the little finger, the adductor and inner half of the flexor brevis pollicis.

Symptoms.—In complete paralysis of the nerve all the above muscles are paralysed and the use of the fingers rendered impossible for many fine movements which require

the lumbricals and interossei. The skin on the back of the little finger, the ring- and half of the middle finger, and on the front of the little finger and half of the next, is anæsthetic. The wrist can be flexed, but the hand falls or deviates to the radial side. The fingers cannot be flexed at the metacarpophalangeal joints or extended at the second and third joints, but some slight power of extension-movement is left in the index and middle fingers, whose lumbricals are supplied by the median nerve. The thumb cannot be adducted, and both adduction and abduction of the fingers are lost more or less completely. Wasting follows, especially in the ball of the little finger and in the interossei, and ultimately the claw-hand seen in progressive muscular atrophy develops; but owing to the median supply of the lumbricals of the first two fingers it is not so typical as the deformity met with in that disease.

Sensory disturbances are common, pain, anæsthesia, and hyperæsthesia being often present along with the motor paralysis. In severe cases the entire cutaneous area of the nerve may be insensible, and trophic changes, as contraction of the palmar fascia, may result. In slight pressure-cases there is no wasting or reaction of degeneration, and recovery is speedy. The prognosis and treatment of the severer cases are identical with those of the secondary neuritis of other nerves.

The *Median Nerve*, arising by two roots—one from the inner and the other from the outer cord of the brachial plexus—receives fibres from all the spinal roots entering into the plexus. It supplies the skin of the palm, of the thumb, and of two and a-half fingers with sensory twigs, and innervates the pronators, the flexors of the carpus, and the long flexors of the fingers, but not the flexor carpi ulnaris (supplied by the ulnar), and only part of the deep flexor of the fingers, also the flexor and abductors of the thumb and the first and second lumbricals.

The nerve is seldom paralysed alone and is very rarely the seat of a primary neuritis, but it may be affected in toxic neuritis, especially in the puerperal type. Nearly all the examples of paralysis of this nerve-trunk are caused by injuries to the forearm. Compression-neuritis has been frequently observed to occur in artificers in metals, as fitters, brass-finishers, locksmiths, &c.

Symptoms.—Where the lesion is severe and high up in the course of the nerve, all the above-named muscles are paralysed, pronation beyond the mid-position is impossible, and the patient, in order to execute this movement, rotates the humerus inwards. When the wrist is flexed the hand deviates to the ulnar side. The second phalanges cannot be bent upon the first ones; flexion of the terminal phalanges of the first and second fingers is impossible, though this

movement can be executed in the ring- and little fingers. The movement of flexion at the metacarpo-phalangeal joints is intact. The thumb is in the position of adduction and extension, and its tip cannot be made to touch the tips of the fingers.

There are usually sensory symptoms present, as often the secondary neuritis is severe; generally the anæsthesia is best marked upon the palmar surface of the hand, which may be completely devoid of tactile sensibility as far inwards as the fourth metacarpal bone. The sensation may be also lost on the posterior aspect of the fingers in the regions supplied by the five digital nerves—*i.e.* the integument over both sides of the thumb, index, middle, and outer side of the ring fingers. There is usually marked wasting of the ball of the thumb.

Combined Paralysis of the nerves of the upper extremity, as already stated, is more common than isolated types of palsy. The entire brachial plexus may be involved in a severe primary perineuritis, in which the fibrous sheaths of the cords and their branches become affected somewhat in the same manner as the involvement of the prolongation of the sacral plexus in ordinary sciatica. This form of neuritis supplies probably the majority of the cases of so-called brachial neuralgia or brachialgia, the symptoms being mainly sensory; the disease is described upon page 1096 under 'Neuralgia.'

Total Plexus Paralysis is nearly always the result of serious trauma, and the symptoms are unmistakable, since they follow such accidents and injuries as fracture of the clavicle, of the head of the humerus, or luxations of this bone; more frequently, however, total plexus paralysis is caused by violent and unskilful attempts at the reduction of dislocations with the heel placed in the axilla; strong traction on the arm may then produce extensive laceration of the cords of the plexus, followed by total palsy of the upper extremity. Even in such a case there may be but slight loss of sensation in the skin over the inside of the arm owing to the irregular distribution of the lateral cutaneous branch of the second intercostal (the intercosto-humeral nerve), which sometimes innervates this region.

The extent and nature of the injury determine the prognosis, which is usually more or less serious, as many cases do not entirely recover the use of the arm, forearm, or hand, wasting following, with deformity and ankylosis of joints from disuse.

There is a type of *partial plexus paralysis* known as Erb's or Duchenne-Erb's Palsy, in which there is a combined paralysis of the shoulder and arm muscles from injuries of the roots of the fifth and sixth cervical nerves or of their corresponding cords in the plexus. The lesion is the result of severe compression of the roots or cords between the clavicle and first rib. It occurs in narcotic sleep in laparo-

tomies and other protracted operations where the arms are misplaced ; it is seen also in hod-men and other burden-carriers, and constitutes the most frequently met-with type of *obstetrical paralysis*, exclusive of the facial palsy, so often seen after forceps-delivery ; it is rare as a primary neuritis.

The deltoid, biceps, brachialis anticus, both supinators, but more frequently the supinator longus, and more rarely the supra- and infraspinatus and subscapularis, are paralysed.

Symptoms.—The deltoid paralysis prevents abduction of the arm, whilst that of the biceps, brachialis anticus, and long supinator prevents flexion at the elbow, so that the arm is kept in the extended position and the forearm and hand in the position of pronation, often whilst the humerus is rotated inwards.

Wasting constantly follows and the reaction of degeneration is present, either complete or partial in extent ; generally there is no anæsthesia observable anywhere ; patches of numb skin may be detected, however, sometimes in various portions of the outer surface of the arm or forearm.

The prognosis depends upon the amount and nature of the injury ; usually recovery is slow. In obstetrical cases the outlook is good if after five or six weeks there is no marked reaction of degeneration and electro-therapy be resorted to early.

PARALYSIS (PERIPHERAL) OF THE NERVES OF THE LOWER EXTREMITY.—If we exclude the sciatic nerve, which is often inflamed primarily in the condition called ‘*sciatica*,’ commonly described as a form of neuralgia, it will be found that peripheral paralysis of the nerves of the lower limb is very much less frequent than paralysis of the nerves of the upper extremity.

LUMBAR PLEXUS.—This plexus, placed in the psoas-muscle, originates in the anterior primary divisions of the four upper lumbar nerves. The plexus or its roots may be diseased in caries of the vertebræ, psoas-abscess, malignant and other tumours ; it may be affected in the ascent of a neuritis from some of its own branches or those of the sacral plexus. Its branches supply the muscles which adduct and flex the hip-joint and those which extend the knee, and they supply cutaneous sensibility to the lower part of the abdomen and front and inside of the limb to the foot.

The *Obturator Nerve*, which supplies the hip and knee joints as well as the thigh-adductors, is not often the seat of an isolated paralysis, but it may be injured during parturition. The main symptom is loss of power in adducting the femur, which cannot be made to cross over its fellow ; the movements of rotation outwards and inwards are also somewhat hindered, and when the hip-joint is flexed the foot is everted. There may be also loss of sensation on the inner

surface of the thigh and leg when the occasional cutaneous nerve is a large branch.

The *Anterior Cruural Nerve* is the largest branch of the plexus, and supplies the skin on the inner side of the thigh and leg as far down as the foot, as well as the knee-joint and all the muscles on the front and outer side of the thigh and half of the pectineus.

The symptoms are loss of power in the extensors of the knee, which soon waste; the knee-jerk is absent; flexion of the hip may be impaired, as when the iliacus is paralysed by injury to the nerve within the abdomen. There is anæsthesia or, in irritative lesions, hyperæsthesia and pain; the loss of sensation in the skin may involve the entire thigh, with the exception of a narrow ribbon-like area along the middle of its posterior surface. The anæsthesia also involves the inner side of the leg and foot and the adjacent sides of the first and second toes.

The *External Cutaneous Nerve* springs from the loop formed between the second and third lumbar nerves and passes out of the abdomen beneath Poupart's ligament near the anterior superior iliac spine. Its position under the firm dense fascia of the thigh, before it gives off its anterior and posterior branches to the skin on the outer side of the hip and thigh as far down as the knee, is of some clinical importance. Being a purely sensory nerve, neuritis of its trunk or branches only causes pain, hyperæsthesia, or anæsthesia; but the indirect motor disturbances are nevertheless rather remarkable, and have led to some confusion and difficulty. Roth described the symptom-complex under the rather misleading title of 'neuralgia paræsthetica.' The writer has closely studied the affection in some well-marked cases in which the following symptoms were evident: Pain of a severe stabbing character was felt along the skin on the outer side of the hip and along the front of the thigh and *occasionally upon the inside* above and close to the knee, where the anterior branch of this nerve sometimes communicates with the internal saphenous nerve. The suffering invariably follows walking or other exercise in which the lower limbs are used, and it soon passes off on resting; it never occurs in bed—*i.e.* there is no *spontaneous* pain. During walking the pain seems to spread to the deeper parts below the fascia. Some of the paroxysms, produced evidently during the compression of the fascia lata by the muscular contractions, may be as severe as a sword-thrust and cause lameness or sudden inability to move. After a mile or less has been covered, the patient, who started with a sound gait and unconscious of any sensory disturbance, hobbles in acute suffering or gives up entirely. Sudden stabs of pain, produced in some slight rotatory movements by the contraction of the thigh-muscles pressing upon the fibrous canal

in the fascia lata in which the anterior branch of the nerve is buried on the front of the thigh, may inhibit the action of the thigh-muscles, and even cause the patient to stumble or fall.

The rapid disappearance of the symptoms after rest is a striking feature in this form of neuritis, and there may be no anæsthesia, though the condition may remain for many years. Where the pain is mainly experienced about the outer side of the hip the case is liable to be mistaken for sciatica, and the writer is satisfied that many cases regarded as of the nature of sciatica are examples of neuritis of the posterior branch of this external cutaneous nerve of the thigh. An important diagnostic point about the value of which there can be little doubt is the ease and freedom from pain by which free movements of the thigh can be executed whilst the patient is lying in bed in the morning. On assuming the erect posture, probably because the entire mass of the thigh-muscles increases somewhat in bulk, these movements may be attended by considerable suffering. The cause is most probably of rheumatic or gouty origin, though the condition has been observed in alcoholic and other forms of multiple neuritis, and it sometimes follows trauma. Rest and anti-rheumatic remedies, especially aspirin, in full doses, give the best prospects of amelioration. The writer has observed a similar type of neuritis affecting the crural branch of the genito-crural nerve after prostatectomy.

SACRAL PLEXUS.—This is formed by the junction of the lumbo-sacral cord (fourth and fifth lumbar nerves) with the anterior divisions of the first three sacral nerves and part of the fourth. From it branches go to supply the obturator internus, pyriformis, gemelli, and quadratus femoris; whilst through its chief branch—the great sciatic—it supplies the muscles of the back of the thigh and all the muscles below the knee. The skin of the buttock and posterior part of the thigh and leg, with the outer part of the latter and nearly all of the skin of the foot, obtain their sensory supply from the branches of the plexus.

The plexus may be involved by pelvic tumours, bone-diseases, and inflammations, and by the pressure of the forceps or foetal head during parturition; but even when such pressure, direct or indirect, is diffuse and extensive it is common for all the branches from the plexus to escape, except those which supply the peronei. The cords of the plexus are not rarely involved in an ascending or in a toxic neuritis; and here also the vulnerability of the peroneal muscles is remarkable.

The *Small Sciatic Nerve*, springing from the lower cords of the sacral plexus, is chiefly sensory in function, supplying the skin of the buttock, back of the thigh, and upper part of the

calf ; but, as it sends a muscular branch to the glutcus maximus, paralysis of the nerve is followed, in addition to anæsthesia of the above regions, by weakness, which shows itself in diminished power of extending the hip and rotating the thigh outwards.

The *Superior Gluteal Nerve*, from the lumbo-sacral cord, is seldom paralysed alone ; when this happens, the weakened medius and minimus are unable to abduct and rotate the thigh inwards, extension movements are diminished, and the gait is oscillating.

The *Great Sciatic Nerve*, which is the continuation downwards of the sacral plexus, is seldom completely paralysed ; more frequently it is the seat of a primary neuritis, or it suffers in the secondary toxic form, especially in gouty, rheumatic, and alcoholic subjects. Under 'Sciatica' its sensory and other symptoms will be detailed, but sometimes the neuritis affects the motor function of the nerve chiefly. When the lesion is a severe one and is high up—*i.e.* near to or in the notch—the leg-flexors and hip-extensors, semi-tendinosus, semi-membranosus, and biceps are paralysed and somewhat wasted along with all the muscles below the knee ; but when the involvement of the trunk occurs lower down—*i.e.* about or above the middle of the femur—the leg and foot muscles suffer, whilst the knee-flexors are unaffected. In these cases the anæsthesia may only involve the skin over the dorsum and sole of the foot ; and though all the leg-muscles are weakened and paralysed, the foot may still be raised in walking by the action of the psoas and iliacus in over-flexing the hip-joint (these muscles are supplied by the anterior crural and lumbar nerves).

The *Peroneal or Musculo-cutaneous Nerve* is a branch of the external popliteal, which is one of the two large divisions in which the sciatic trunk terminates. The trunk of the external popliteal is generally involved with its peroneal branches, in which case the following muscles will suffer from loss of power—*viz.* the peroneus longus and brevis, tibialis anticus, extensor longus digitorum, and extensor brevis digitorum. The muscles being unable to flex the ankle, the foot falls or drops by its own weight, and the first phalanges of the toes cannot be extended, so that the characteristic 'drop-foot' corresponding to the 'drop-wrist' is produced. The *steppage gait* is marked, the entire limb having to be raised high in order to prevent the toes catching upon the ground. Contractures are liable to follow, so that permanent talipes equinus supervenes, and the unopposed interossei may permanently flex the first row of phalanges. There is cutaneous anæsthesia of the front of the leg at its outer aspect and on the dorsal region of the foot. The muscles waste and trophic changes may be present. This

condition of paralysis is common in the various forms of multiple neuritis ; in that produced by lead, often the tibialis anticus escapes in the same remarkable manner in which the supinator longus in the arm remains unaffected in many cases.

The *Internal Popliteal Nerve* innervates all the muscles at the back of the leg and sole of the foot, and furnishes sensibility to the sole and plantar surface of the toes, as well as to a variable portion of the skin on the front of the leg. When completely paralysed, the plantar flexion of the foot and toes is impossible and the flexed leg cannot be rotated inwards. Contractures of the unopposed muscles cause a permanent talipes calcaneus, and the first phalanges are persistently over-extended, or even sub-luxated, whilst the two distal ones are flexed, so that a 'claw' or griffin-like deformity of the digits follows, preventing the patient springing from the toes as in ordinary walking. There is anæsthesia of the sole and outer border of the foot and of the plantar aspect of the toes.

The *Plantar Nerves* are seldom affected alone ; but in multiple toxic neuritis they are commonly involved, the anæsthesia, hyperæsthesia, and motor disturbances varying considerably in different cases, as already described under 'Neuritis.'

The prognosis and treatment of the peripheral palsies met with in the lower extremity differ in no way from those affecting the upper extremity.

PARALYSIS, PSEUDO-HYPERTROPHIC.

(See under MUSCLE-ATROPHY, p. 1060.)

PARALYSIS, SPASTIC

Loss of motor power in paralysed muscles shows itself in three forms—the *simple* or *functional*, the *flaccid*, and the *spastic*. In the first, when complete, there is simply loss of movement ; the muscles fail to respond to the will and the affected limb falls by the force of gravity if raised by passive motion, as seen in hysteria. When the loss of power is less marked, the paresis is of the same type : there is no obvious change in the weakened muscles, save that they fail to respond to volition, or do so with feebleness ; this form rapidly passes into the next type. In flaccid paralysis, as the name implies, there is obvious loss of muscular tonicity ; the affected limb is limp, lax, or flabby, and there is no resistance to passive motion : this form of paralysis is recognised as the *spinal* type. The spastic type of paralysis is characterised by the presence of tonic spasm ; there is increased tonicity, showing itself in muscular rigidity and stiffness or resistance when passive movement of the limb is attempted.

This condition depends upon disease of the neurons in some portion of the upper motor tract. This latter consists of the dendrites of the cortical motor cells, of the axis-cylinders which proceed downwards from these, and of their arborisations as they end in terminal brushes in the anterior cornua of the cord. The spastic variety of paralysis, it must always be remembered, is of the true *centric* or *cerebral* type, as distinguished from the *spinal*. It may be caused by disease or injury of any one or all of the three constituents of the upper neuron. Thus in hemiplegia of cerebral origin the spastic form of paralysis is met with, whether the disease be in the cortical cells or in the axis-cylinders converging to form the internal capsule or other portion of the motor path. (See description of the entire motor path on page 1212.)

As the axis-cylinders of the upper neurons pass downwards, forming the pyramidal tracts running in the lateral columns of the cord, their disease in the cord also will cause this cerebral or spastic type of paralysis; hence, though a spinal disease, the type of the paralysis must nevertheless be regarded as *centric* or *cerebral*.

With the increased tonicity which eventuates in spasm and often in permanent contractures, other features are present; thus there is great increase or excess of myotatic irritability, as seen in exaggerated knee-jerks and ankle-clonus, probably because the inhibitory or controlling influence of the upper neurons is withdrawn from the lower ones. Though the muscles are weakened or paralysed, they do not waste or degenerate, because they are not cut off from the trophic influence of the nerve-cells of the lower neurons located in the anterior horns; hence also they do not present the reaction of degeneration. In all these features spastic or centric paralysis is in marked contrast to flaccid or spinal paralysis, with its wasted and degenerated muscles, which fail to respond to faradism whilst still contracting to the continuous current; and it is also accompanied by diminution or loss of the reflexes. Confusion must be guarded against in the conception of *spinal* paralysis, as in the case of the cerebral type, by remembering that the spinal type may be seen in its typical state in disease of the nuclei of the bulbar nerves in the brain, the cord being intact. It is also seen in multiple peripheral neuritis without cord-lesions; in its most marked form it is met with in poliomyelitis anterior acuta, in all which instances the paralysis is the result of disease of some portion of the lower motor neurons. This type is also seen in an imperfect form in the muscular dystrophies (pseudo-hypertrophic paralysis), in which there is no reaction of degeneration, and atrophy is concealed by apparent enlargement. (The spinal and cerebral types of palsy are seen in a *mixed* form where the disease simultaneously involves the

motor cells of the lower neurons and the axis-cylinders of the upper ones in the pyramidal tracts, constituting Amyotrophic Lateral Sclerosis in which wasting and spasticity exist together (page 1067).

Spastic paralysis is usually seen in both the lower limbs, and hence the term is often used as a synonym for spastic paraplegia; but paraplegia with spastic signs may result from compression-myelitis, chronic or transverse myelitis, spinal meningitis, disseminated sclerosis, Erb's syphilitic disease of the cord, ataxic paraplegia, primary combined sclerosis, Little's disease, hereditary spastic spinal disease, general paralysis of the insane, hysteria, &c., as well as from primary lateral sclerosis. It must be always remembered that any disease or injury of the pyramidal tract may cause degenerative changes below the lesion, ending in a secondary spastic paralysis. The spastic paralysis caused by primary disease of the pyramidal tract will be described in the following article.

PARAPLEGIA, SPASTIC, PRIMARY (Primary Lateral Sclerosis)

As implied by its title, this is a disease whose main clinical symptoms show themselves at first by loss of power in both legs, with spasticity and increase of reflexes, but without muscular wasting or degenerative reaction. Its synonym, 'Primary Lateral Sclerosis,' implies that its pathology is that of a primary isolated degenerative sclerotic change in the lateral columns, especially in the crossed pyramidal tracts.

Etiology.—The disease affects men and women in about the same percentage of frequency, generally after the age of twenty and before forty. A neurotic family-history is common, and the exciting causes have been traced in several cases to exposure to cold, trauma as in falls, syphilis, toxæmias as in plumbism, the puerperal state, and infectious fevers, and in a few cases to arthritic diseases of the lower extremities.

Symptoms.—The disease begins in the lower extremities in an insidious manner, the patient complaining for months of being easily tired on walking moderate distances. With the gradual loss of power is always associated a sensation of stiffness or rigidity which interferes with rapid and delicate movements of the legs and feet; this is often for a considerable period more marked than the paralytic phenomena, and is usually more severe in one limb than in the other.

The typical spastic condition described in the previous article steadily develops; the superficial and deep reflexes and all forms of reflex action are exaggerated. Ankle-clonus is well marked, motor power is considerably diminished, the muscles are not wasted and do not show the reaction of degeneration, and sensation is unimpaired. Finally, but perhaps not till after the expiration of some years, both limbs become so involved by the persistent tonic extensor-spasm

that walking becomes extremely difficult or impossible, as the patient is unable to raise the toes or flex the knees, so that the limbs are dragged behind the body or pulled forwards as if the joints were encased in splints. It may require extreme passive force to flex the knee owing to the extensor-spasm, but when this has been accomplished the spasm disappears, to immediately appear again on the commencement of extension, as if the patient had suddenly executed a brisk kicking movement (clasp-knife rigidity). Permanent contractures may supervene, especially in bed-ridden cases. Any flexor movement may excite a severe paroxysm of tonic spasm, which occasionally may pass into the clonic type. The arms in some cases pass through the same gradual process, but often they escape; very occasionally the trunk-muscles are involved, and the masticators may suffer. Rarely are the sphincters or pupils affected; when they are involved they form no part of the clinical picture of pure spastic paraplegia, but, like many other complications seen in cases of paraplegia accompanied by spasticity, they belong to the original condition of which the spastic paralysis is a secondary event.

There is an *hereditary family-type* of the disease, described by Strümpell, in males, usually showing itself only after the twentieth year; but in these cases, which are extremely chronic in their duration, sensory symptoms are present and the sphincters suffer; the majority of them are not pure examples of primary lateral sclerosis.

The type of spastic paraplegia known as *Little's Disease* is noticed as commencing soon after birth or after the first year of life, when the patient should be able to walk. The gait is characteristically spastic; the thighs are adducted and rotated inwards, sometimes cross-legged progression resulting. The heels are raised, the knees and hips slightly flexed, and the child gets along by dragging the spastic limb after him while the opposite one rests on the toes. The arms are often involved in a somewhat similar manner. This type of spastic paraplegia really belongs to the birth-palsies caused by meningeal cerebral hæmorrhage; it usually follows severe deliveries, and is associated generally with mental deficiency, speech-disturbances, squint, cerebral convulsions, and athetosis, though many authorities maintain that some of the cases are examples of a spastic paraplegia of isolated spinal origin in the pyramidal tracts.

Primary spastic paraplegia is of very slow course, may exist for many years, and has no tendency to shorten life.

Pathology.—There have been but a very few autopsies on pure cases of the primary disease, and some authorities are still doubtful if such cases ever appear; but there cannot be a doubt about the spastic symptoms as above described being due to a primary degeneration or isolated sclerosis of

the crossed pyramidal tracts as they run in the lateral columns of the cord. Other changes observed at the same time in the other tracts or columns may be either secondary or coincident.

Diagnosis.—It must be always insisted upon that by far the greater number of all the cases of paraplegia or of paresis of the lower extremities which show spastic symptoms are not cases of *primary lateral sclerosis*. The diagnosis of the primary disease can only be justified after the demonstration of the absence of all other symptoms indicative of spinal or cerebral disease, the one feature of typical spastic paralysis alone constituting the clinical picture of lateral primary sclerosis, or, as it is forcibly put by Oppenheim, 'every symptom which does not clearly belong to the spastic-paretic symptom-complex is an index of another disease.' The diagnosis will therefore only be possible when the other symptoms characteristic of the different diseases mentioned at the end of the preceding article are eliminated (see p. 1227).

The slow onset of the disease puts out of question the cases of myelitis and other cord-diseases which begin more or less acutely.

Ataxic paraplegia is differentiated by the presence of loss of incoordinating power and by the instability of the gait, the presence of Romberg's sign, and generally also by some sensory disturbances, as the disease in its typical form is the result of a combined degeneration of the posterior columns and of the pyramidal tracts.

Hysterical paraplegia is of more or less sudden onset, and the peculiar extensor character of the spasm (lessening with flexion) is of itself sufficient to differentiate spastic paraplegia from all functional affections; and, moreover, in simple hysteria a true ankle-clonus is not obtainable, and there are always sensory symptoms.

Birth-palsies are eliminated by the history of the onset and by the usual absence of mental deficiencies. Primary disease of the spine with spastic paralysis in children practically never occurs, unless the lesion producing it be of cerebral origin or unless there be caries of the vertebral bodies.

The prognosis as regards recovery is practically hopeless, but it is within the bounds of possibility that the condition might stand still or cease to be progressive. The prognosis as regards life is satisfactory; the ailment has no tendency to shorten life.

Treatment.—This is unsatisfactory. Rest is essential whilst the disease is passing through one of its spells or phases of getting obviously worse, as may be observed by increased severity in the extensor spasmodic seizures. Massage of the gentlest and mild passive movements should be assiduously persevered in, and all real fatigue avoided. Drugs are

powerless to arrest the degenerative action, and strychnine may make matters worse ; the same may be said of electricity.

The congenital or juvenile type, which is, however, very rarely, if ever, a pure lateral sclerosis, may be much assisted by massage and gymnastic exercises. When contractures occur, unless the loss of power be very marked, they should be relieved by tenotomy and the use of splints ; occasionally more radical measures, as myotomy, transplantation of tendons, and other orthopædic measures may be resorted to.

PARAPLEGIA, ATAXIC

This is a form of spastic paraplegia, combined with ataxic signs, caused by a degenerative sclerosis of both the pyramidal tracts and the posterior columns of the cord. The symptom-complex is often described under the title of Combined Lateral and Posterior Sclerosis.

Etiology.—Nothing is known definitely of the cause of this rather uncommon malady, which is very seldom hereditary. It occurs at about the same period of life in which spastic paraplegia first appears ; the exciting agents—cold, prolonged muscular exertion, trauma, sexual excesses, and pernicious anæmia—have been supposed to be etiological factors. Certainly syphilis is not answerable for the disease, save in such a small percentage of cases as might be accounted for by mere coincidence.

Symptoms.—The onset of the affection is most gradual and insidious, and when the gait has become markedly influenced by the established disease the symptoms may be observed to be as just stated—a combination of those of locomotor ataxia and spastic paraplegia. The ataxia is sometimes at first more marked than the loss of motor power, but in the later stages the opposite condition prevails. The type of the symptoms depends upon the degree of intensity with which the degenerative process has attacked either of the involved tracts. Thus when the pyramidal tract suffers most and early, as is usually the case, the spastic symptoms preponderate.

The onset will resemble that of the previously described primary lateral sclerosis, which shows itself usually by some rigidity, accompanied by a tired feeling in the legs. The loss of power may not be noticed or complained of by the patient, but his gait becomes unsteady and he is likely to fall when walking in the dark or upon attempting to turn suddenly. He soon finds that he cannot stand with his feet together when his eyes are shut. At this stage his condition is hardly likely to be mistaken for tabes, as the spasticity is well marked, and if careful examination be made there will always be found

loss of motor power, without sensory or eye symptoms; but the chief distinctive feature is the increased myotatic irritability, as seen in the *exaggerated knee-jerks* and often in the presence of ankle-clonus. Gradually other tabetic symptoms may be added on to the incoördination, as lightning-pains, bladder-weakness, Argyll-Robertson pupils, &c.; these are rare, however, usually never appearing at any stage. The arms are also often involved, but generally never to the same extent as the legs; sometimes tremulous or ataxic movements are noticeable in the facial muscles.

In the late stages of the paralysis, according to Gowers, who first thoroughly described the disease, the ataxic symptoms fall into the background as the motor weakness increases and passes into an almost typical picture of primary lateral sclerosis, without sensory or mental symptoms of any kind.

Occasionally, however, mental failure or exaltation like that seen in general paralysis supervenes, and ultimately the bladder becomes atonic. When the degeneration of the posterior columns is progressive and reaches the lumbar enlargement, the exaggeration of the knee-jerks may disappear and the tabetic sign of total loss of this reflex may become developed.

Ataxic paraplegia pursues the same chronic course as lateral sclerosis, and shows little tendency to markedly shorten life, though occasionally cases are recorded where death has supervened inside a few years. Where an earlier fatal issue is reported the cases are probably examples of a different disease—*i.e.* of a sub-acute form of combined lateral and posterior column disease, not of systemic distribution, but of inflammatory origin, in which secondary degenerative changes may possibly have followed later on. This type of disease, though possessing marked ataxic and paraplegic features, commences with the sensory and motor symptoms of a diffuse myelitis or of an acute or sub-acute disseminated encephalo-myelitis; it is usually associated with anæmias of the pernicious type, or it follows the infectious fevers, ulcerative endocarditis, &c. The writer has seen this form of typical ataxic paraplegia follow influenza and run an acute course, ending in recovery. In the common chronic degenerative form of the disease recovery probably never occurs.

There is a *combined primary sclerosis*, known as Putnam's Disease, whose pathological anatomy has been investigated by Risien Russell, Dana, Collier, and others. This affection usually runs a sub-acute course, and these authorities maintain that, though not a system-disease in the true sense, there is a disseminated *degeneration* involving numerous small areas in the posterior and lateral columns. The disease is often associated with pernicious anæmia, and is probably of toxæmic origin.

The signs are those of Gowers' ataxic paraplegia plus sensory symptoms, as anæsthesia, numbness, tingling, &c., the condition finally passing into a flaccid palsy of the limbs, with loss of knee-jerks and involvement of the sphincters; emaciation and often mental symptoms appear a short time before death.

Pathological Anatomy.—Little need be said upon this, as the pathology of the disease is implied in its name of combined lateral and posterior sclerosis. The degeneration is of the usual sclerotic type, resembling that seen in tabes; but Gowers contrasts its distribution with that of the sclerosis of tabes, and points out that in ataxic paraplegia the change is often less marked in the lumbar region, and that the root-zone of the postero-lateral column is much less deeply involved. In the lateral columns, according to the same authority, the sclerosis is often not strictly systemic in character, inasmuch as it does not strictly limit itself to a single system of fibres, though the pyramidal tracts are chiefly involved—*i.e.* the morbid process often extends beyond the limit of the tract chiefly affected. The grey matter and membranes are not involved in the degenerative process.

Diagnosis.—The typical symptoms of a lateral sclerosis with increased knee-jerks, combined with loss of incoördination, in a disease of chronic course and insidious onset where sensory symptoms are absent, leave little doubt about the diagnosis, and exclude ordinary *tabes*, in which the knee-jerks are always absent. In *primary lateral sclerosis* there are never ataxic symptoms.

Disseminated cerebro-spinal sclerosis is eliminated by the absence of the characteristic scanning speech and of optic-disc atrophy.

Syphilis of the spinal cord may cause somewhat similar symptoms, but there will always be a history of a comparatively recent infection. The predominance of sensory symptoms, as severe girdle or neuralgic pains caused by the meningeal inflammation, which is the main pathological feature in spinal syphilis, is almost distinctive. There is a form of paraplegia, known as *Erb's syphilitic spinal paralysis*, which is easily differentiated, since the symptoms are mainly those of a considerable loss of motor power with a less degree of spasticity, though the reflexes are greatly exaggerated. The general features resemble those of a chronic transverse myelitis. In this symptom-complex of Erb the sensory symptoms are slight, the sphincters suffer, and ataxic symptoms do not form a part of the clinical picture. Though the onset is gradual the condition is not progressive, and often ends in recovery.

Myelitis is recognised by its acute or sub-acute onset; but in the rare cases of ataxic paraplegia already referred to, which

are supposed to be of inflammatory origin, the differential diagnosis may be impossible till the steadily progressive nature of the true degenerative ataxic paraplegia has had time to show itself.

Lathyrism or *Lupinosis* is a disease produced by the use of various varieties of lathyrus or vetch employed in India and North Africa (Algiers) for mixing with wheaten flour to make bread. The main symptom is that of a more or less typical spastic paraplegia, in which the arms always escape. The condition may sometimes be identical with primary lateral sclerosis, only the history and the endemic distribution of the disease settle the diagnosis of this toxic form of sclerosis of the pyramidal tract. The condition is allied to pellagra, described upon page 1236.

The prognosis of ataxic paraplegia has been dealt with in the description of its progress, and little can be said of its treatment, as the disease is beyond the reach of drugs.

PARAPLEGIA, SIMPLE

Under this heading for convenience may be placed the paralytic conditions in which both lower extremities are involved as the result of inflammatory diseases of the cord—*i.e.* myelitis in its different forms, as already described under 'Myelitis,' p. 1077, and compression-myelitis produced by vertebral disease. Inflammations of the spinal membranes may also produce paraplegia. The different forms of meningeal disease are described upon page 1038, with syphilitic paraplegia.

Alcoholic, Hysterical, and other paraplegias will be found under the headings of the affections in which they occur.

Simple Senile Paraplegia is a condition first noticed by Gowers as occurring in elderly subjects. The chief feature in the affection is a weakness in both lower extremities, which gradually progresses without wasting and sensory symptoms or alterations in the reflexes. In a few cases the arms and face become affected, which suggested to Gowers that the condition was allied to or identical with those cases of paralysis agitans in which a tremor never developed.

PEDICULOSIS, or PHTHIRIASIS

This is the name given to the condition of the body when infested with lice. It is always caused by or associated with neglect and filthy or unclean habits which interfere with or prevent the necessary ablutions of the body, so that after an accidental invasion of the individual's person or of his raiment the parasites are permitted to increase and multiply

without hindrance. Three different varieties of louse are found on the human body :

Pediculus capitis infests the hair of the scalp and lays its numerous eggs in small conical baskets, glued to the hair-shaft, usually close to the skin ; but before the development of the ovum is accomplished the embryo and its envelope (usually known as a 'nit') may be hoisted some distance from the level of the scalp by the growth of the hair-shaft during the eight or ten days of incubation. The female measures about a line in length, the male considerably less ; the colour varies with the skin of their host, but more so with the colour and nature of the filth which is permitted to accumulate on the scalp, being usually yellowish-grey in tint when found in the white races ; it may be identified by its triangular head and by its thorax, which is shorter than that of the body-louse.

Pediculus corporis, or *P. vestimentorum*—the body-louse—is considerably larger, with an oval head, stronger legs, and larger abdomen. Its colour is yellowish-white, with a dark margin ; it lives in the creases of the body-linen and derives its nourishment from its victim's blood, as the head-louse does, by suction after biting. In the act of feeding it inserts, as described by Morris, a membranous tube into the opening of a sweat-duct, through which it draws up the blood, at the same time depositing some irritating secretion. The puncture is marked by a minute red speck, caused by the hæmorrhage into the gland-duct, and is surrounded with a variably sized œdematous areola. In some individuals who are very sensitive to the poisonous secretion of the parasite the areola may be extensive and resemble a small urticarial wheal.

Pediculus pubis, as its name implies, infests the hair over the pubes, and is usually transferred from one individual to another in sexual intercourse. It may afterwards migrate to the hair of the eyebrows, beard, or axilla. The parasite is flatter, broader, and shorter than the other varieties, and is of a uniform brownish-yellow colour ; owing to its shape, and general resemblance to the common crab, it is usually known as the crab-louse. Its bite is often followed by peculiar sub-cuticular bluish or slate-grey discolorations (maculæ ceruleæ or taches bleuâtres), which are supposed to be caused by the inoculation of a pigmented secretion.

The bite of all three varieties of these parasites is followed by a variable amount of itching, so that multiple inflammatory lesions are produced by their irritation, the finger-nails causing linear abrasions in scratching and inoculating various germs which are often found on the surface of the skin. Eczema, impetigo, crythema, and dermatitis of varying degrees of intensity are thus the outcome of the excoriation

and irritation caused by scratching, which also usually produces hæmorrhages. The products of this traumatic dermatitis dry up and form scabs or crusts, so that, especially on the scalp, a disgusting matted condition of the hair is found, which may appear as a large crust or cake over the occipital region, known as *plica polonica*, which may be swarming with the vermin and their ova, and which is often associated with enlargement of the cervical glands.

Upon the body the secondary inflammatory papular and eczematous lesions caused by continual scratching may lead to a considerable brawny or leathery thickening of the skin and to the deposition of much generally diffused pigment, which, with the linear excoriations and inflamed papules, constitutes the so-called 'vagabond's disease' so commonly met with in tramps.

The crab-louse causes similar irritative lesions through the intense itching and consequent scratching arising from their presence on the pubic region; more frequently its traumatic lesion is purely eczematous in type.

Diagnosis.—The discovery of the lice or their nits in the hair of the scalp, or of the parasites in the creases of the body-linen, easily establishes the diagnosis. In the pubic-louse infection, inspection at once confirms the diagnosis.

The bright red spot, with its darker central punctum, which results from the bite of the common flea (*Pulex irritans*), and the similar punctum surrounded by a large wheal or zone of erythematous infiltration caused by the bite of the bed-bug (*Cimex lectularius*), are easily differentiated.

In the diagnosis of the skin-lesions seen in pediculosis too much importance is not to be attached to the linear form of the excoriations produced by the finger-nails. Minute inspection will reveal numerous papillæ whose inflamed tops have been mowed down in the scratching, but both these markings are seen in lichen and in every skin-disease or condition associated with itching; only the discovery of the minute specks caused by the bites of the parasite or the presence of the parasites themselves will settle the diagnosis.

Treatment.—Absolute cleanliness is a thorough preventive. When the parasites have infested the head the speediest way is to cut off all the hair when this has become matted. In milder cases sponging with strong vinegar will dissolve the cement by which the nits are attached to the hair-shaft, when fine combing will remove them along with the parasites themselves. If the hair be saturated with common paraffin-oil before combing the same result is obtained, but, in addition to mechanical removal, those parasites which escape the comb perish by the oil.

After the removal of the hair, the crusts, impetigo, &c., should be treated by warm boric-acid poultices, followed by

a mild carbolie or white precipitate ointment or the use of a cerate containing powdered stavesacre-seeds.

Where the body is infected the individual's clothing should be baked in an ordinary drying-oven or disinfecting chamber, whilst he should himself be thoroughly cleansed in a warm bath of permanganate-solution.

Crab-lice, as a rule, are easily killed by ordinary white precipitate ointment or a weak mercurial preparation smeared over the skin of the pubic region without removal of the hair.

PELLAGRA

This is a condition caused by the use of diseased maize, or maize which has got mixed with some vegetable-poison or which has undergone fermentation before being employed as food. It is met with amongst the poor in Roumania, in Italy, and regions bordering on the north of the Mediterranean. The symptoms are mainly those of a more or less typical ataxic paraplegia, and the anatomical lesion consists of a combined sclerotic degeneration of the posterior and lateral columns of the cord, with atrophy of the large cells in the anterior horns, and often with much thickening of the pia mater.

In addition to gastro-intestinal symptoms, which are most variable, there is nearly always present a severe erythematous eruption, accompanied with much burning pain, followed by desquamation and pigmentation. The disease is a chronic one, and, as the erythema returns each year, the subsequent thickening with pigmentation of the skin becomes very marked, but gradually it gives way to atrophy.

At the end of three or four years, by which time the ataxic paraplegia has become well established, the patient gradually wastes, and often mental symptoms become prominent, with suicidal tendencies, and he sinks into a comatose condition usually by the end of the fifth or sixth year. Where the condition is recognised early the outlook is more hopeful when the cause is remedied and the environment of the patient altered. The greater proportion of the victims are middle-aged females. The condition is allied to Lathyrism, described upon page 1233.

The disease is also allied etiologically to ergotism, and the erythematous eruption closely resembles that seen in *Acrodynia*, a disease caused by some unknown food-poison, and seen occasionally in the soldiers of the army of France. The erythema is followed, as in pellagra, by exfoliation, thickening, and pigmentation, and occasionally paraplegic symptoms supervene, which, however, are probably the result of a multiple neuritis, as the sensory symptoms in the palms and

soles are well marked in most cases. Recovery after some weeks is the common result, a fatal issue, except in debilitated senile patients, being extremely rare.

The treatment of pellagra is obvious: the food-supply should be changed immediately and the environment altered; if possible, the patient should be removed from the locality in which he has become infected.

PEMPHIGUS

This is a disease characterised by the presence, on previously healthy skin, of an eruption consisting of large blebs, caused by circumscribed elevations of the epidermis containing liquid. The mere existence of bullæ or blebs does not constitute pemphigus, since these appear in various other skin-affections, as in cheiropompholyx, syphilis, hydroa, impetigo, eczema and scabies, and in traumatic conditions, as the result of scalds, blisters, &c. Ormerod therefore defines pemphigus as 'a disease in which, without obvious cause and upon apparently healthy skin, an eruption arises, which from first to last consists of bullæ.

Several varieties of pemphigus have been described; these may be, however, limited to three distinct or definite types—*i.e.* *P. vulgaris*, *P. foliaceus*, and *P. vegetans*.

PEMPHIGUS VULGARIS

Symptoms.—The onset of the disease is sudden, the first thing observable being a crop of blebs, bullæ, or very large vesicles resembling blisters, filled with clear, pale, amber liquid. These are discovered upon any part of the body or limbs, and may reach their full dimensions within twenty-four hours after being first noticed; when unusually large they sometimes measure as much as one inch across. When first seen, the skin immediately surrounding each bleb is usually quite normal in colour, but after a day or two a slight erythematous zone of pink immediately engirdles each bulla; about the same time the contents begin to show some degree of opacity, turbidity, or even purulency.

The bullæ come out in crops, each crop lasting but three or four days, and they may be scattered all over the body by successive additions to those originally appearing on the first day of the onset. After the contents have become opaque, the blebs, which are usually tense and plump, burst or shrivel and dry up, forming crusts; these spontaneously fall off, leaving a purple spot of skin covered by new epidermis, which in a week or more has become distinctly pigmented. In a few weeks more the brown pigmented spot begins to fade, and eventually merges into the healthy skin without

any line of demarcation. It is not unusual to see crops of bullæ in different stages of desiccation, surrounded here and there by the erythema or pigmentation caused by the crop which has already disappeared.

When the blebs burst extensive excoriations may result, and a slowly healing but very superficial ulcer may mark the site of the base of each ; but as a rule, in mild cases, this only occurs where the bullæ have been injured by friction or scratching.

The mucous membranes may also be the seat of the eruption, the eye, nose, mouth, and throat being involved ; rarely the trachea and bronchi are affected, but the vagina, and even the intestinal surface have suffered sometimes. A certain degree of fever, usually of mild type, may be noticed at the onset ; it is, however, often so slight as to be overlooked, but it may return at a later stage when the raw excoriated surface becomes infected with the pus-forming and other organisms usually present on the surface of the skin.

In some severe cases the contents of the bullæ are distinctly hæmorrhagic, and in others, which become purulent, sloughing of the skin may follow. Often the first sign of the termination of the disease is the appearance of one or two solitary bullæ when a crop was expected. There are sometimes serious constitutional weakness and nerve-prostration, though the local irritation of the eruption is seldom very marked, and sometimes itching is very slight ; but occasionally scratching may produce secondary lesions, and apparently assist the spread of the eruption to different parts of the body.

The progress of the disease is slow, owing to the number of recurrent attacks, and the patient may die from exhaustion after many months, or even years, in which there may be numerous recurrences, characterised by successive crops of bullæ appearing at variable intervals.

Such is the usual course of *chronic* pemphigus, the most common type of *P. vulgaris* ; but not rarely an *acute* form of the disease is met with, which is ushered in with rigors, a high temperature, delirium, grave prostration, mental and physical, with uræmic symptoms and albuminuria. There is often a history of a poisoned wound in these cases, many of which occur amongst butchers. The bullæ appear in such rapid succession that the crop-like periodicity of the more chronic form is not observed, the entire area of the skin being thickly studded over with bullæ appearing in rapid and continuous succession. The fever keeps up, rapid wasting accompanies the exhaustion, and death may occur within a period of three or four days ; more usually the fatal termination in these acute cases is deferred for two or three weeks, and in some of the early fatal cases it is noteworthy that the temperature has not been elevated. A mild form of the

disease, known as *pemphigus neonatorum*, occurs in new-born infants, and sometimes assumes an epidemic type amongst ill-cared for children in the practice of an uncleanly midwife. This form appears to be of the nature of an impetigo contagiosa.

Etiology and Pathology.—Little is known of the causes which produce the disease. It is more common in young children than in adults, but it may occur at any age. A few cases have been recorded in which heredity played an obvious part. The acute form has long been suspected to be of microbic origin, and many observers have isolated organisms supposed to be specific. Pernet has recently examined the bullæ in a case supposed to have been caused by sewer-gas, and obtained pure cultures of *bacillus pyocyaneus*. Saundby has found the *bacillus coli* in the fluid from the blebs. Eustis obtained a diplococcus from the fluid in the blebs, cultures of which, when injected into the veins of a rabbit, caused death. The rabbit's blood yielded the diplococcus, and the organism, when injected into the pig, caused a pustular eruption. Bowen believes that those cases of acute pemphigus, following wounds, are caused by an attenuated form of the same micro-organism which produces foot-and-mouth disease. Michélan and Cassaet consider the disease is caused by retention of chlorides in the blood.

From the frequency with which vesicles and bullæ are found in neuritis and cord-disease, the chronic variety of pemphigus has been supposed to be of neuritic origin—a trophoneurosis, or, as held by Morris, of angioneurotic nature. There is no evidence that the disease is really contagious in the ordinary sense of the word, but a contagious form, according to W. Turner, has been met with in India.

The anatomical changes in pemphigus vulgaris are similar to those found in any acute inflammations of the skin. There is exudation of serous fluid from the vessels of the papillæ, which splits the rete layer, the upper part of which, with the horny layer, forms the roof of the bulla, and on puncture of a bulla it collapses like a blister, there being no loculi. As regards the pathology of the rarer forms of pemphigus nothing is known.

PEMPHIGUS FOLIACEUS

Symptoms.—This rare form differs materially from *P. vulgaris* in its progress and severity. The bullæ are always more or less flaccid or wrinkled, and never present the tense or plump appearance of those seen in the former variety. Their contents are turbid, sometimes bloody, and often semi-purulent; they burst easily or dry up into yellow crusts, under which extensive red excoriated patches of skin are found. The sticky purulent liquid clings about the old

collapsed walls of the bullæ, and gradually undermines the adjoining epidermic layer of the hitherto healthy skin. Ultimately large and extensive areas of the skin become ulcerated, which seldom completely heal; these may cause unsightly deformities. Crops or groups of isolated imperfectly formed bullæ continue to appear from time to time in the neighbourhood of the red exuding surface, till the greater part of the skin of the body is involved in the ulcerating process. The disease causes wasting and intense misery from the pain of the excoriations, though there is little itching or fever, and it ultimately ends invariably in death from exhaustion or marasmus. It has been known to supervene upon long-continued attacks of the common form. The mucous surfaces are commonly involved, and the larger bronchi not rarely invaded. The course is very chronic; in one case seen by the writer it had lasted for seven years, and probably this patient did not finally succumb till two or three years later.

PEMPHIGUS VEGETANS

Symptoms.—This is a still rarer variety, and differs from the former types in several essential features. Thus the bullæ, which are small and resemble ordinary pemphigus in appearance, after bursting leave an excoriated and ulcerated base from which warty outgrowths spring up, so that some authorities regard the condition as a distinctly different disease. It first shows itself upon the mucous membrane of the vulva, mouth or pharynx, but later on bullæ and papular lesions appear upon the extremities and trunk, which coalesce by serpiginous ulceration till large areas are involved by the warty or papillary growths. In a case recently described by Duckworth the affected regions became pigmented, of a slate-blue colour, and the adjacent uninvolved areas of skin were deeply stained by brown pigment, the patient's appearance suggesting a leprous ulceration. The warty raised granulations are usually surrounded by a ring of small bullæ, and, especially in moist or warm regions like the arm-pit or around the genitals and anus, these exude a highly offensive and foul secretion, not unlike that observed in deep burns affecting a cachectic patient.

Death occurs almost invariably, and the fatal issue is seldom protracted beyond a few months, unless the ulceration of the mouth and fauces is not so severe and extensive as to prevent swallowing, when life may be prolonged for several months.

Diagnosis.—*Pemphigus vulgaris* is not likely to be confounded with any other skin-affection if due care be taken to observe that *the bullæ spring from healthy or uninjured skin*. The presence of other lesions, in which large vesicles or blebs

of serum may appear as adventitious elevations of the skin, will distinguish such diseases as *urticaria*, *erythema*, *eczema*, and *ecthyma*; moreover, the highly characteristic feature in pemphigus of the bullæ appearing in successive crops belongs to no other cutaneous affection.

Syphilis is the only other disease, according to Payne, in which bullæ appear upon otherwise healthy or uninjured skin, the condition being sometimes described as syphilitic pemphigus; but even then it much more closely resembles *P. foliaceus* or *vegetans* than *P. vulgaris*. In the first place, the rarity of the former two conditions may well be allowed to weigh in the differential diagnosis; secondly, both these affections are progressive, and, finally, fatal, whilst syphilitic pemphigus is amenable to remedies. The condylomata of syphilis have a thickened border and smooth surface, contrasting markedly with the warty surface of *P. foliaceus*, surrounded by a ring of small bullæ. The history of a comparatively recent infection and signs of other syphilitic lesions will settle the diagnosis in most cases. There is a so-called *epidermolysis* or *congenital traumatic pemphigus*, which shows itself in a remarkable weakness of the skin, by which trivial injuries and frictions are followed by bullæ; it is not a true pemphigus, but seems allied to hæmophilia.

Hydroa is differentiated by the polymorphous character of the eruption as described under 'Dermatitis Herpetiformis,' p. 267.

Prognosis.—In *P. vulgaris*, in very young subjects, the outlook as regards the danger to life is very favourable, unless the attack be of the typical acute variety. Since many patients suffer from recurrence of the disease, the forecast in this respect is always doubtful. In old subjects of weak and debilitated habit pemphigus is always a serious disease, and the longer it has lasted the less likely is it ever to pass completely away.

P. foliaceus, though practically always a fatal disease, can have its slow course still further retarded by suitable treatment.

P. vegetans, as already stated, is invariably fatal, and the gravest prognosis may be confidently made as soon as the diagnosis is once established, though the lesions may at first seem to be of trifling degree.

P. neonatorum, if of syphilitic origin, is very often fatal.

Treatment.—The drug upon which most reliance is to be placed, and which should always be administered at once as a routine in every case, is arsenic in full doses. In young subjects its effects are striking, and bear out Mr. Hutchinson's first reports upon its great value in this affection. The local treatment is at the best palliative, but much may be done in the way of diminishing the extreme misery of the victim

of *P. foliaceus* by permitting him to spend as much time as possible in a suitable warm or tepid bath, and by the thoroughness with which his bullæ are protected from injury and the excoriations dressed with mild antiseptic ointments.

PERICARDITIS, ACUTE

Inflammation of the pericardial sac can scarcely be regarded as a primary disease, being almost invariably the secondary result of some microbic infection, rheumatic or otherwise.

Etiology.—The great majority of all cases of pericarditis capable of being diagnosed during life, whether acute or chronic, are of rheumatic origin. The disease may appear in its typical form when the joint-affection has been so slight as almost to escape notice. This is more likely to occur in children, and it can hardly be doubted that most, if not all, cases of so called *primary* or *idiopathic* pericarditis occurring in early life are examples of rheumatic origin—a view which is rather strengthened by the facts that endocardial trouble almost invariably accompanies the pericardial mischief, and is seen in choreic subjects in whom no rheumatic history is obtainable. Of patients suffering from rheumatic fever Osler's figures show that about 6 per cent. have pericardial inflammation; Sibson pointed out that the complication was much more frequent in those young subjects whose occupation had previously subjected them to severe labour. Males are much oftener affected than females, and a large percentage of the pericarditis occurring in rheumatic women is observed amongst hard-working young domestic servants. The disease in rare cases may precede the joint-inflammation; probably it begins during the first week of the illness in rheumatic-fever patients, though not noticeable till a later period.

Septic diseases, as pyæmia, erysipelas, acute necrosis of bone, puerperal peritonitis, and puerperal fever are sometimes primary etiological factors. The disease is not rare in other microbic infections, as tuberculosis, influenza, scarlatina, measles, and pneumonia.

Bright's disease accounts for a considerable proportion of cases, but as it is often a terminal event in this affection its causation is of less clinical importance, latent pericardial disease being frequently met with at the autopsy in granular kidney.

Extension of the inflammatory action to the pericardium in acute pleuritis and empyema is a not uncommon complication. This is to a considerable extent the explanation of the frequency with which acute pneumonia (especially when affecting the left lung) is associated with pericarditis, though

in some cases of severe pneumonia doubtless the infection is through the blood-stream. Mediastinal suppuration and even abdominal mischief by penetrating the diaphragm may excite pericarditis; aneurismal, hydatid, malignant, and tubercular growths in the vicinity of the pericardium may act likewise either by extension of the inflammatory processes set up by them or by the direct perforation of the sac by the contents of an abscess-cavity. Trauma, even without perforating wounds or fracture of ribs may excite pericarditis.

Morbid Anatomy.—The changes vary with the stage of the inflammatory action, and from a study of cases examined at all periods the following are observed. First, a stage of congestion or increased vascularity is observed, differing in no way from the early changes seen in other serous inflammations. This is rapidly followed by the fibrinous exudation-stage, as seen in cases where both changes may be observed to occur at the same time in different parts of the pericardial sac. A layer of fibrin, of whitish colour, and often of firm consistency is found spread over the interior of the sac and over the visceral layer enveloping the heart. It varies in thickness from the thinnest film to that of an elastic firm coating, half an inch or more in depth; usually at first it can be readily peeled off from the inflamed opaque and thickened membrane, though at a later stage, after partial organisation of its layers, it can only be detached by some forcible scraping. When the sac is opened, the appearance to the naked eye of this sheet of fibrinous exudation is striking and unmistakable. Owing to the movements of the heart in its systole and diastole, the layers of sticky exudation are alternately pressed together and drawn from each other, so that what is usually described as a honeycombed or reticulated surface is formed on the opposing surfaces of the pericardial sac. At other times it presents a villous surface, which has appeared to the writer to closely resemble the fleecy wool upon a lamb's back, whilst again distinct shaginess may be caused by the fibrin being drawn out in long irregular shreds. Bands of tough, firm, elastic lymph or fibrin may stretch across between the visceral and parietal layers, and even loculi may be thus formed.

More or less fluid is present, which may be either free in the sac or lodged in the fibrinous interlacements or loculated spaces in the exudation; but occasionally a firm dry exudation alone is present, in which case the adhesions are abundant.

In the ordinary cases of 'pericarditis with effusion' there is a considerable quantity of transparent amber or greenish-yellow fibrino-serous fluid poured out between the layers. This may vary from 5 to 10 oz. or more in children, to 30 or 60 oz. in adults. In the pericarditis caused by tubercular

deposits, Bright's disease, empyema, and septic fevers the fluid may consist of pus alone or pus mixed with serum, whilst in purpura and cancer there is often much blood present. As the fluid is removed from the sac, usually large quantities of fibrinous flocculi or gelatinous masses are found to occupy the dependent parts. Sometimes, when the effusion is great, the solid exudation on the layers of the pericardium is slight; but as a rule in rheumatic cases there is a considerable thickness of fibrinous exudation, though some parts of the sac are more involved than others.

When the flocculent or leathery membrane is scraped off with a knife or with the finger-nail, the visceral layer of the pericardium is found to be opaque, thickened, and congested, and the underlying heart-muscle, for a varying depth, is seen to be the seat of a myocarditis which, under the microscope, reveals itself by granular and fatty changes in the muscle-cells.

More or less endocardial inflammation is also present, as described under 'Endocarditis'; this is probably very seldom an extension from without, being the result of a concurrent endocarditis, but occasionally the heart-muscle has been found involved in its entire thickness in chronic purulent cases in which the resulting endocarditis may then be the outcome of a simple extension of the inflammatory action arising in the pericardium. Changes of an inflammatory or degenerative kind may be also detected in the coats of the great vessels within the sac. In chronic cases especially, the outer surface of the pericardium may be covered with fibrinous deposit, which will then also involve the contiguous pleura. Bacteriological examination of the fluid often reveals the presence of the specific micro-organisms of the primary disease, as streptococci, staphylococci, pneumococci, bacilli of tubercle, bacillus coli, and diplococcus rheumaticus.

Symptoms.—The disease being essentially a secondary one, the symptoms in the early stages will be those of the primary rheumatic fever, pneumonia, septic infection, &c. In pericarditis without much effusion symptoms may be entirely absent throughout, the latency of the affection being in a fair percentage of cases complete; but signs of great diagnostic value are usually found present when carefully sought after.

Pain is often present, but it is most variable in character, site, and intensity; it may be severe in dry or so-called plastic pericarditis, and entirely absent in acute extensive effusions. It is common to find a sharp stabbing pain in the early stage, which disappears when fluid has distended the sac and separated the inflamed surfaces. The precordial region or the epigastrium, close to the xiphoid cartilage, may be the site of tenderness, amounting to marked hyperæsthesia, with or

without spontaneous, dull, continuous pain. When effusion has occurred to any marked degree a sense of great tightness and oppression, aggravated by deep inspiration, is often experienced, but occasionally a large effusion may produce no subjective symptom as long as the patient lies still in bed. It is therefore necessary to make daily routine examinations in rheumatic fever, especially since the pain of the joints compels the patient to remain absolutely immovable. Upon changing the posture, flushing of the face alternating with pallor, pain, palpitation, dyspnoea, rapid pulse, with irregularity or intermittence and marked weakness, cough, aphonia, and even dysphagia, may become suddenly prominent, though not previously noticeable. All these symptoms may be present, however, whilst the patient is quite still, when the sac is full. The pressure of the distended sac may cause such embarrassment of the ventricular contractions that the pulse-wave at the wrist may entirely disappear towards the end of inspiration, constituting the *pulsus paradoxus*; this sometimes is most marked on the left side.

Sometimes the radial presents characters similar to the water-hammer pulse of aortic regurgitation. As the embarrassment due to pressure increases, lividity, cyanosis, and anasarca appear, the veins of the neck become engorged, orthopnoea is established, and death from asphyxia threatens.

Nervous symptoms show themselves in insomnia, mental excitement, and restlessness associated with delirium and twitchings; occasionally stupor and a tendency to coma, or a profound melancholic depression may be marked.

The temperature is a variable quantity, and little can be said of it, since almost always the primary disease is associated with some pyrexia. When this is not the case, however, the temperature, even after the supervention of pericarditis, has been sometimes found to remain normal. Notwithstanding the last-mentioned fact, if in rheumatic fever, or other condition likely to be the cause of pericarditis, the temperature should suddenly spring to a high point, and especially should the pyrexia be associated with sudden delirium, the cardiac region should be at once examined for the physical signs indicative of pericarditis.

Physical Signs.—Various types of the disease have been described, the classification being made upon the basis of the amount of fibrino-serous fluid present. Thus, when this is absent, the condition is spoken of as a *Plastic Pericarditis*, and the after-stage of this variety or type is described as *Chronic Adhesive Pericarditis*. The usual form, in which a considerable amount of effusion is poured out, is styled *Fibrino-serous Pericarditis*, or *Pericarditis with Effusion*. The nature of the effusion has given rise to such names as *purulent* and *hæmorrhagic* pericarditis.

The physical signs of the plastic type may be regarded for clinical purposes as corresponding to the early signs observed in cases in which effusion afterwards is poured out, and may be considered as common to both.

Inspection.—This, in the early stage, will reveal nothing characteristic, save in rare cases some irregularity in the rhythm of the apex-beat.

Palpation.—The finger-tips placed over the precordial region in many cases will convey a distinct sensation of a *thrill* or *friction-fremitus*, which feels as if produced immediately under the tissues palpated and not deep in the heart itself. Its site varies; usually it is felt over the middle of the right ventricle. It is limited in extent and differs from an endocardial thrill in its abruptness and in not conveying a distinct shock to the finger-tips or palm of the hand, though systolic in time. It is obviously produced by the rubbing together of the fibrinous layers on the opposed surfaces of the pericardial sac during the systole of the ventricles.

Auscultation.—A pericardial *friction-sound* is audible, often most intense over the front of the heart about the site of the thrill, and, like the latter, it is caused by the grating together of the fibrin-clad surfaces, giving the impression of being produced immediately beneath the stethoscope, and it is usually intensified by pressure. It is a double or to-and-fro shuffling sound, heard both during systole and diastole, though very occasionally a single systolic rub may alone be detected. The sound is of very variable quality—sometimes soft and rubbing, whilst at others it may be of a harsh and grating character, likened to the creaking of new sole-leather. The two portions of the sound, separated by the silence, are usually of about the same length, and do not correspond strictly in duration or in rhythm to either of the true cardiac sounds; they may both be distinctly audible, especially after the cessation of the second sound.

When very loud and harsh, this to-and-fro murmur may be heard all over the front of the heart and along the edge of the sternum, in which case its intensity may entirely silence the normal cardiac sounds. Its abrupt termination conveys no idea of a blowing sound, and, though usually somewhat sharply circumscribed in its site, it is never ingravescent, like the typical auriculo-systolic murmur, and, moreover, it has no definite line of conduction or transmission, such as is characteristic of mitral or aortic murmurs.

When listened to for a few minutes, its relations to the normal sounds may be noticed to alter, and sometimes the second sound may be heard to ring out distinct from the second part of the murmur. When the fibrinous exudation is very limited it may entirely disappear on changing the posture of the body, if some little fluid be present; it is sometimes

minimised, but usually it is augmented, in intensity by deep inspiration or expiration, and, unlike a pleuritic friction-rub, it is not silenced by suspended breathing. As already stated, the pressure of the stethoscope renders it more distinct, and, as pointed out by Roberts, this test may change every characteristic feature of the murmur—its duration, rhythm, quality, pitch, and site, and may cause the rub to double if it has been single. These distinguishing points in the pericardial murmur, taken together with its variability from day to day, constitute a physical sign which, when present, may be regarded as practically pathognomonic of pericarditis. The murmur often *does not disappear when fluid is effused*, even in quantity; it may become more intense, but its site is then changed to a higher level, and it sometimes continues all through the stage of effusion in marked contrast to the friction-sound produced by pleuritis.

Signs of Effusion.—No alteration in the cardiac dulness is observable in acute cases till the advent of fluid effusion. When this occurs to any marked extent, *inspection*, before resorting to percussion, will reveal some bulging of the sternum, left costal cartilages, ribs, and intercostal spaces. Ewart draws attention to the elevation of the left clavicle, which is raised by the effusion so that the inner half of the first rib may be palpated. Bulging of the epigastrium may be obvious, and even fluctuation may be obscurely appreciated in this region just below and to the left of the sternum. The liver is depressed, and the respiratory movements on the left side diminished. The impulse of the heart against the chest-wall is seen at a higher level, usually in the fourth, but often in the third space, and to the left of the nipple-line. This impulse is seen and felt to be diffuse, and is not the true punctate apex-beat, but the weak thrust of the middle of the right ventricle against the chest-wall. In some cases, from the beginning, an undulatory impulse may be visible but not palpable, and this is the rule in the later stages of effusion when, owing to the progressive weakness of the ventricular contractions, the impulse entirely disappears and the muffled cardiac sounds are only heard with difficulty.

Percussion in the stage of effusion will reveal marked changes in the extent and shape of the outline of cardiac dulness. The dull area is pyramidal or pear-shaped, with the base below and the apex of the pyramid or triangle above. In extensive but not extreme distension of the pericardial sac with fluid, the narrow upper portion of the dulness rises beyond the level of the third costal cartilages, and the lower limit of the base of the pyramid will extend as far downwards as the lower margin of the sixth rib. As this area is percussed from the clear lateral regions of the thorax on each side in an inward direction, the dulness will be found to

be very marked and to extend on the right side as far outwards as the parasternal line—*i.e.* to a line midway between the edge of the sternum and the line of the right nipple. Upon the left the widest part of the dull area will be found to extend about half-way between the left nipple-line and the anterior axillary line.

In cases of extreme distension, the upper blunt end of the cone may reach as high as the top of the sternum, and the base-line may be as low as the level of the upper part of the seventh interspace, whilst from side to side the limits of this line may reach from the right nipple-line to almost as far as the left mid-axillary line. Except at the very margins, the degree of dulness is *absolute*, since the distended sac, pushing the lungs aside, comes into direct contact with the sternum and anterior thoracic wall. In the earlier stages of pericardial effusion Rotch's sign may be observed—*i.e.* dulness on the *right side* over the space between the fifth and sixth costal cartilages, where the right margin of the heart forms an angle with the liver. From the dulness caused by dilatation of the heart the differential diagnosis by percussion alone may be difficult; but an important point is the determination of the absence of resonance on the left side well beyond where the true cardiac apex may be located by careful and exact auscultation.

In extreme effusion a small dull patch may be found behind, about the angle of the left scapula, caused by compression of the lung, and over it tubular breathing and ægophony may be well marked, especially in thin young subjects. Sometimes, according to Ewart, tubular breathing may be audible in front below the right breast in the nipple-line.

The effects of changes in the patient's posture may confirm the results of the percussion-signs; thus sometimes the sitting position causes an elevation of the dulness, and a change to the right side may cause the left margin of dulness to shift somewhat inwards, and if there has been tubular breathing over the compressed margin of the left lung it may then disappear entirely after deep inspiration. The prone position likewise may cause the dulness and ægophony at the angle of the scapula to vanish. Important information may be obtained by changes of posture as regards the location of the apex of the heart in some cases.

The use of the X-rays may confirm the extent of the effusion and the degree of displacement of organs.

The progress of pericarditis with effusion varies within wide limits; in acute cases the area of dulness may be demonstrated as increasing markedly within twenty-four hours. Not rarely, from the first sign of the disease till the appearance of evidence that absorption has commenced, only three or four days may elapse, and all the signs of effusion

may have passed away before the termination of a week. More frequently, however, two or three weeks are requisite for this complete resolution. Except in the septic and hæmorrhagic cases, recovery may be expected with tolerable certainty, the disease seldom proving immediately fatal; but the possibility of the ultimate consequences following adhesion of the visceral and parietal layers of the sac should nevertheless place the affection in the category of serious diseases. Extensive effusions may remain in exceptional cases at a standstill, and pass into a chronic condition, as in some examples of acute pleuritis.

Complications, as pleural effusion especially, are not uncommon, and a co-existing or secondary endocarditis may seriously modify the course and symptoms of the disease.

Diagnosis.—The chief points have been already dwelt upon in the description of the *physical signs*; for it must always be remembered that *subjective symptoms* must count for little in a disease so prone to run its entire course in a truly latent manner. The affection is more likely to be entirely overlooked in the case of children, especially where the arthritic manifestations of acute rheumatism are not obviously manifest. The daily exploration of the cardiac area in such cases should never be omitted. The first appearance of a to-and-fro friction-rub may be taken as conclusive evidence of a fibrinous exudation; the percussion and other signs will determine from day to day whether the affection is to remain of the plastic type throughout or be followed by serous effusion. The latter condition, as already emphasised, seldom entirely obliterates the friction-murmur, even when the distension of the sac is great. The presence of some slight pleural effusion at the same time—which is, moreover, a rather common event—may obscure the diagnosis, but only when the liability to this complication is forgotten.

Hydropericardium, when the inflammatory or constitutional disturbances are absent or latent, is likely to be suspected or accepted; but the differential diagnosis is easy when the absence of the friction-rub is demonstrated and the primary cause of the dropsy of the pericardial sac is discovered—viz. chronic Bright's disease or valvular affection in its last stages. Moreover, in these cases the amount of fluid in the pericardium is never extensive, and some can always be detected in both pleuræ and in the peritoneal cavity, and general anasarca is also certain to be present.

Acute dilatation of the heart may be mistaken for pericarditis with effusion, but the careful search for the cause of the dilatation will always reveal the nature of the case. The diffuse and wavy character of the impulse, with the visible apex-beat displaced downwards and outwards, the clear though sometimes feeble first sound, and the absence of the triangular

shape of the area of dulness and the absence of the to-and-fro friction-murmur, are usually of themselves quite sufficient to eliminate pericardial effusion. Moreover, the physical signs do not vary on changing the posture, and the symptoms of cardiac embarrassment are always more serious and urgent.

Endocarditis is often present at the same time that the pericardial sac has become involved, but endocarditis never gives rise to the friction-fremitus or produces the characteristic to-and-fro murmur; when effusion has already occurred pericarditis should not be mistaken for the endocardial affection.

Pleural effusion is not likely to be mistaken for pericardial effusion, unless where the presence of old pleural adhesions limits the fluid exudate to the neighbourhood of the cardiac area. A pleuro-pericardial friction-sound will disappear on the outpouring of the fluid; there will be displacement of the heart to the right, and the base of the thorax behind will be dull on percussion. Concurrent pericarditis and extensive pleural effusion can only be recognised by the signs of pericardial distension remaining after aspiration of the pleural sac.

Concurrent *pneumonia* may cause the dulness of a distended pericardium to be overlooked; the diagnosis of the double inflammation must then rest largely upon the discovery of the to-and-fro murmur, which should be differentiated from a pleural friction-sound caused by the cardiac impulse. The condition should be suspected when the dyspnœa and cardiac embarrassment are greater than might be expected from the pulmonary signs.

Prognosis.—As already stated, the immediate prospects of recovery are good in the great majority of cases of rheumatic origin, and this is true even when some endocarditis co-exists. In infants and aged subjects, however, the condition is sometimes fatal even when there is no evidence of other than a rheumatic origin. The most unfavourable signs are those which indicate serious involvement of the cardiac muscle, as determined by the pulse and irregularity of the ventricular contractions. Hyperpyrexia is almost invariably a sign of a fatal issue when the pericardium has been previously involved.

Septic and pyæmic cases are almost always fatal, the prognosis being that of the primary disease.

The gravity of the situation is accentuated when the effusion becomes so extensive as to cause dyspnœa, with cyanosis. Death may result from asphyxia or from a sudden attack of syncope induced by some abrupt change from the lying to the sitting posture; more rarely a large effusion may terminate fatally after reducing the patient to a state of profound asthenia by a prolonged course of suffering. The use of prompt measures for the removal of the fluid alters greatly the prognosis when the grave symptoms are the result of pressure

upon the heart and lung; it has little influence upon the outlook in the rapid acute cases of extensive effusion with profound nervous prostration, delirium, &c. The ultimate prognosis as regards the secondary effects of pericardial adhesions cannot be arrived at till after the expiration of a considerable period.

Treatment.—The first indication is to be found in the primary disease; thus in rheumatic cases the routine administration of salicylates is the best line of treatment. The dangerous depressant action of moderate doses of the drug upon the cardiac muscle (when it is observed) is almost certainly due to impurities. Sulphocarbolates and quinine in septic cases may be given, but little is to be hoped from their action when a purulent exudation has already occurred. Rest is essential—as nearly as possible to absolute repose of body and mind. The inclination of the patient regarding his posture may be obeyed within certain limits, the best position being the horizontal, with the shoulders slightly raised. Pain, if present, may be relieved by opiates, leeching, ice, or a small blister applied over the centre of the precordial area, and this latter agent may also assist in promoting absorption of fluid if applied at a later stage.

As soon as the quantity of fluid poured out becomes so extensive as to produce a very marked difficulty of breathing, the pericardial sac should be aspirated, the best position for the puncture being in the left fifth interspace, near to the sternum. As soon as a reasonable suspicion of the purulent nature of the sac-contents is formed, it is wise to verify it by a hypodermic needle, after which free incision should be performed by any of the methods described in the author's 'Dictionary of Treatment,' pp. 679-681. Only in cases where the pyopericardium is associated with a manifestly fatal malady, as in pyæmia, in ulcerative endocarditis, or in advanced malignant disease of the sac, should the patient be denied the hope that active surgical procedures may afford.

PERICARDITIS, CHRONIC

This term is practically synonymous with '*Adherent Pericardium*' or *Chronic Adhesive Pericarditis*, though sometimes the stage of effusion in serous pericarditis may be indefinitely prolonged into a chronic pericarditis in which the sac remains distended by the liquid products of the original fibrino-serous inflammation.

Etiology and Pathology.—The condition may be the immediate result of an acute attack of plastic pericarditis or of pericarditis with effusion, in which case the etiological factors will be those causing these affections, as already

detailed. It is more likely to supervene in cases where the effusion was purulent, if the patient survive the acute attack. More frequently, however, adherent pericardium is the result of a latent chronic inflammation which develops insidiously or commences by a series of recurring attacks of dry plastic inflammation long after the absorption of the fluid poured out in an acute pericarditis.

Pleural or other chronic inflammatory mischief arising in the lung outside the sac may gradually extend inwards and glue together all the structures in the mediastinum, and even involve both layers of the pericardium and thoracic wall. In many advanced cases, discovered only at the autopsy, the history may give no clue to the etiology of the disease, an acute attack of pericarditis never having been recognised at any time in the patient's life. Sometimes tubercle and new-growths are the obvious causes ; and some authorities recognise a congenital variety, probably the result of the rheumatic process before birth.

Morbid Anatomy.—All grades of adhesion are met with, from isolated fibrinous bands stretching between the visceral and parietal layers of the sac to a partial or absolutely complete obliteration of the space. The uniting or obliterating layer may be a dense fibrous envelope upwards of half an inch in thickness, encircling the entire heart ; and in advanced cases it may become more or less completely calcified by the deposition of lime-salts in the fibroid tissue constituting its structure. Sometimes the outer surface of the pericardium is normal and free from adhesions, whilst the interior of the sac is obliterated ; in other cases the inner surfaces of the sac may be normal, its outer layer being glued to the pleura and chest-wall by its involvement in an old adhesive mediastinitis. The most typical class of case is that in which the internal adhesions between the visceral and parietal layers of the sac are found in association with the results of an external pericarditis, gluing and matting together the outer layer of the sac with the left pleura and front of the chest-wall.

The changes in the heart itself are of great interest and importance, though even when the pericardial sac has been entirely obliterated the heart in rare cases may show no anatomical change. In a large proportion of cases, however, considerable hypertrophy or dilatation, or a combination of both conditions, is found, even when no valvular lesion co-exists ; and the degree of the enlargement is always most marked in the class of case where internal pericarditis has been associated with the external variety. The cause of the hypertrophy and dilatation is doubtful : it probably is the result of a combination of factors, such as myocardial change, increased labour in carrying on the work of the circulation,

and the presence of valvular disease in a considerable proportion of the cases. It must always be remembered, however, that not only does adherent pericardium occur with neither hypertrophy nor dilatation, but it is sometimes associated with wasting of the cardiac muscle.

The bulk of the enlargement is usually to be accounted for by the thickening of the walls of the right ventricle; the auricles are generally little changed.

Symptoms.—The condition is often entirely latent, being only rendered evident at the autopsy. Even when suspicion is aroused by the history of a previous severe attack of pericarditis there may be neither symptoms nor signs of the disease to warrant a diagnosis. As might be inferred from this fact, when signs and symptoms are present they may vary immensely in different examples of the condition, scarcely two cases being alike. The following symptoms have been observed in typical cases, though usually only a proportion of them may be found in any individual example of the disease.

Pain.—This may be of a dragging character, and aggravated by deep inspiration or by anything which excites the action of the heart. It commonly comes on at times in severe angina-like paroxysms, associated often with great *palpitation*. *Dyspnoea* is not uncommon, alone or in combination with lividity or cyanosis; *anasarca* and *ascites*, together with hepatic and splenic enlargement and albuminuria, are seen as in failing compensation from valvular and other causes. Thus the main symptoms are such as are commonly present in all cases of cardiac hypertrophy associated with dilatation and failure of compensation, though the distinction is made by Broadbent that right-ventricle failure in adherent pericardium is often associated with pallor, sometimes without dyspnoea and often without pulmonary engorgement.

Physical Signs.—Certain signs are often found present when carefully looked for, some of which have been regarded as almost pathognomonic.

Inspection.—The usual bulging of the precordial region seen in all cases of great cardiac enlargement will be obvious; very occasionally the opposite condition is observed in which the parietes are depressed or contracted.

The impulse of the heart varies greatly in character in different cases, but as a rule in a typical example it extends over a much wider area than the normal and over a wider area than that observed in ordinary cardiac hypertrophy. The entire precordial area may be elevated from the level of the second to that of the sixth space, and from beyond the right edge of the sternum to beyond the left nipple. The impulse, though greatly diffused, may be weak and undulatory. The apex-beat often cannot be accurately located; its

most characteristic feature is its fixation often in a slightly elevated site, and if steadily watched the tissues over it are seen to retract during or immediately after systole. This *apical recession* or retraction is a valuable sign; it is often associated with a recession of a considerable portion of the precordial area, especially of the intercostal spaces close to the sternum on the left side; the lower end of the sternum may also recede in systole. These two latter signs may be marked even when no apex-beat or apical recession is visible.

A sign of perhaps still greater importance when present is the *retraction of the lower ribs* in the lateral and posterior aspect of the left thorax visible during systole. Broadbent, who attached great importance to this phenomenon, explained its production upon the theory that it was due to the diaphragmatic attachments to the two floating ribs (eleventh and twelfth) and to the contiguous asternal ribs, these attachments being dragged upon during systole owing to the extensive external adhesions between the heart and a large portion of the upper surface of the diaphragm, and probably also between the heart and the thoracic wall. These adhesions also prevent the descent of the diaphragm, so that in inspiration the epigastrium does not bulge and the respiratory movements on the left side of the thorax are diminished.

Another sign of some value is obtained by inspection during deep respiratory movements, when usually the apex of the heart will be found to be fixed and immovable; this may also be corroborated by altering the posture, when a similar fixity will be demonstrated. Collapse of the veins in the neck is occasionally seen to occur in diastole, and is supposed to be due to the suction-power of the heart in diastole; it occurs at the moment of the diastolic shock to be immediately referred to.

Palpation.—Many of the inspection-signs may be corroborated by the hand placed over the precordial region. The most important sign to be determined by this method is the presence of a distinct diastolic rebound or shock immediately following the systolic recession. Unfortunately it is often absent, but when present it is conclusive evidence of adherent pericardium. It is felt over the different areas where the systolic recession is marked, and its stroke is often more vigorous and forcible than the systole. The finger on the radial pulse will often find the wave to disappear at the end of inspiration—the *pulsus paradoxus*.

Percussion.—The area of cardiac dulness is usually markedly increased; it corresponds to the enlargement of the heart, and has no other significance. The dulness is often absolute, owing to the adhesion of the outer part of the pericardium to the front of the chest-wall, which pushes the collapsed lung well to the side. It is of some value when the area is not

altered in extent or shape by deep respiratory movements or by changes of posture, and, moreover, the dulness is likely to be prolonged nearer to the top of the sternum than in ordinary cardiac hypertrophy.

Auscultation.—These signs are most variable and uncertain, and are, moreover, liable to be confused with the murmurs produced by the valvular lesions, with which the pericarditis is so frequently associated. A special presystolic murmur has been described as due to the adhesion, but it is impossible to be certain of its significance, if present, since such murmur may be caused by mitral narrowing. Of more importance is the absence of an accentuated pulmonary second sound, so constantly present in old valvular lesions. A harsh to-and-fro murmur is very occasionally audible, since the condition is liable to be associated with recurrent attacks of otherwise latent inflammatory action in a sac which has only been partially obliterated. F. T. Roberts emphasises the important fact that, owing to the heart being fixed to the chest-wall, the sounds may be remarkably superficial in character, and in posterior pericardial adhesion they may be heard with great distinctness behind.

Diagnosis.—The presence of any one sign, save that which is caused by the recession of the lower false ribs or the diastolic shock felt immediately succeeding the systole, is not sufficient for diagnosis; but all the other signs, or a number of them taken together, will usually justify a diagnosis. Of great importance in the survey of the case is the recognition of marked failure in the power of the right ventricle, as seen in dyspnœa, anasarca, albuminuria, ascites, and hepatic enlargement, without the presence of mitral lesions. Marked polycythæmia has been several times observed.

Prognosis.—The condition of a patient in whom the signs manifestly justify a diagnosis must always be considered as grave. The gravity depends upon the inevitable changes which the muscular tissue of the heart ultimately must undergo when the circulation is so seriously handicapped, though for a long time the functions of the organ may be carried on without marked symptoms. The degree of gravity may be gauged by the amount of failure in the compensatory changes, as in other cases of broken compensation, and the treatment is identical.

PERICARDIUM, DROPSY OF

This condition is also called 'Hydropericardium,' both of which names define it and distinguish its passive serous effusion from the fibrino-serous exudation poured out as the result of the inflammatory process in pericarditis.

Etiology.—Fluid in the pericardial sac in small amount is very commonly found in the autopsies of many diseases, being probably a *post-mortem* or immediately *ante-mortem* transudation of serous fluid from the vessels; such exudations obviously are not of any clinical importance. The distension of the sac during life is never great; it is generally found as part of the general anasarca which occurs in Bright's disease or in the late stages of failure of compensation following valvular lesions. Occasionally it may be the result of aneurismal or other growths which press upon the veins of the pericardium or interfere with the cardiac veins. In rare cases an acute form of dropsical distension of the sac has been observed to occur in scarlatina, acute nephritis, and in various purpuric blood-conditions, and, more rarely still, in pulmonary diseases and in acute mediastinitis.

Symptoms and Signs.—These are such as have already been described under 'Pericarditis' as distinctive of accumulation of fluid in the sac, but the characteristic to-and-fro murmur, pain, feverishness, and all evidence of inflammatory action are absent. Symptoms are only present when the distension of the sac is so great as to cause dyspnoea; and this is aggravated by the accumulation of dropsical fluid, which invariably occurs in both pleural cavities and in the peritoneal sac.

The fluid differs in no respect from that found in the other serous cavities in cases of general dropsy. It is an alkaline amber-coloured liquid of similar composition to blood-serum.

The prognosis and treatment are those of the primary renal or valvular disease; and as the pericardial distension is usually a terminal event, the outlook is always grave, and tapping of the sac is seldom indicated.

HÆMOPERICARDIUM.—Blood in the pericardial sac is not a rare complication of pericarditis with effusion, especially when this has been secondary to malignant growths.

Etiology.—A sudden outpouring of blood into the pericardial sac may occur, independent of preceding pericardial inflammation, as the result of punctured wounds or perforation of the gullet by the angular projection of dental plates which have been accidentally swallowed. It occurs sometimes with startling suddenness in purpura and in scurvy and from rupture of cardiac or aortic aneurisms.

Symptoms.—These may be only such as are indicative of the shock, sudden collapse, pallor, and syncope usually present in all cases of severe internal hæmorrhage, rapidly ending in death. When the opening through which the blood has gained access to the sac has been a small one the shock is less marked, and the symptoms of cardiac and respiratory embarrassment, with pain, have time to develop for several

hours before death; the percussion-signs of pericardial effusion may be then obvious.

The diagnosis will, however, only be possible in most cases when the antecedent condition has been recognised.

The prognosis is always grave, and, except in rare traumatic cases where surgical measures are indicated, it is absolutely hopeless.

PNEUMOPERICARDIUM.—Gas in the pericardial sac is never found except in association with serous effusion, pus, or blood, as its presence always is associated with a rapidly developed pericardial effusion.

Etiology.—The etiological factors which permit the admission of blood between the layers of the pericardium are also usually present, as wounds, perforation of hollow air-containing cavities in gangrene, and pulmonary abscess-cavities, gastric ulcer, or perforation of the œsophagus. Sometimes the gas has been the result of rapid decomposition of the contents of a pyopericardium. Though this is held by most authorities to be a *post-mortem* change, the writer once observed it during life in a patient suffering from a large pyæmic abscess in the knee-joint. Air has sometimes been admitted accidentally in aspirating the pericardium.

Symptoms and Signs.—There may be bulging over the precordial region on inspection. Palpation will reveal a very weak apex-beat or the absence of a punctate impulse, though there may be a diffuse flickering during systole felt over the middle of the right ventricle. Occasionally when the palm of the hand is laid flat upon the chest the observer may appreciate a sensation not unlike that experienced whilst palpating a dilated stomach when active fermentation is going on or when borborygmi are present in the small intestine.

Percussion will demonstrate a dull and also a hyper-resonant area, which change places on altering the posture of the patient; the tympany is usually accompanied by a metallic quality. These signs, like the following, will obviously vary greatly in proportion to the amounts of gas and liquid which are present. Auscultation reveals metallic or bell-like sounds of a very remarkable and generally unmistakable kind, as splashing, gurgling, or churning of the liquid is effected by the cardiac contractions and relaxations. These sounds have been described as resembling the noise made by a slowly revolving water-wheel. They may be exaggerated by shaking the patient, when the loud bell-like first sound of the heart will ring out clear during the splashing. Occasionally a to-and-fro murmur may be detected, with some metallic quality added on. The splashing sounds may sometimes be heard at a considerable distance from the patient.

Diagnosis.—It must be remembered that in the absence of symptoms of cardiac embarrassment (and such absence is

the rule), the above symptoms and rhythmic sounds may be closely simulated during cardiac systole by the agitation of a cavity in the lung which contains air and fluid when this is in close proximity to the heart. The metallic tinkling and succussion-sounds heard in pneumohydrothorax are easily differentiated when the other obvious signs of that condition are taken into consideration. Gas in a dilated stomach in association with a large amount of liquid may give rise to somewhat similar auscultatory phenomena, but these disappear immediately after the passage of a tube.

The prognosis is always most grave, but when aspiration or a free opening is clearly indicated in traumatic cases the patient should have the faint chance which such operations sometimes afford.

PYOPERICARDIUM has already been referred to under 'Pericarditis'; it is usually associated with septic conditions, as pyæmia and the various septicæmias which sometimes accompany the exanthematous fevers and ulcerative endocarditis. It is not rarely observed to complicate suppurative mediastinitis, and has sometimes been produced by the rupture of a gangrenous or phthisical cavity in the lung or by the extension of the suppurative process in virulent empyemas or purulent peritonitis. Coutts has observed purulent pericarditis frequently in children dying from empyema, and emphasises the necessity for a careful examination of the heart in this condition in all young subjects.

Its diagnosis, prognosis, and treatment have been already referred to under 'Pericarditis.'

PERITONEUM, DISEASES OF

These are in the majority of cases of an inflammatory nature, and are dealt with under the succeeding headings of Peritonitis, Acute, Chronic, and Tubercular. Fluid effusions constituting ascites are described under 'Ascites,' 'Liver, Cirrhosis of,' &c.

CANCER OF THE PERITONEUM

Etiology and Pathology.—Cancer in this region is rarely *primary*, but secondary deposits in the membrane are very common in malignant disease which has originated in the viscera of the abdomen or pelvis. The rare primary growths may be practically regarded as of an epithelial type, though their histological structure shows that the cells are endothelial, arising from the endothelial cells of the serous surface and of the lymphatic vessels. Their cells occur in clusters or alveoli of connective tissue following the course of the lymphatics, and hence by some pathologists are strictly considered as of

the nature of an alveolar or endothelial sarcoma. The growths may be diffused over the surface of the peritoneal membrane in small tubercles, which are not unlikely to be mistaken for ordinary miliary tubercle; or they may be found in large masses located in the great omentum, forming a tumour of considerable dimensions, resembling that often observed in chronic tubercular peritonitis.

Colloid cancer, when found in the peritoneum, is almost certainly of secondary origin, though the primary source may be undiscoverable, as the tumour may reach enormous dimensions and involve the entire sac and all the organs in the abdomen. Any type of malignant tumour may occur in the peritoneum as a secondary growth in cancer of the liver, stomach, ovaries, &c.

Symptoms.—In the rare diffuse primary form the symptoms are those caused by interference with the function of the bowel by the remarkable shortening of the mesenteric folds, which, as already referred to, may produce the peculiar retraction of the umbilicus described upon page 81. Where the deposit occurs in large nodular masses in the omentum these may be felt through the abdominal wall, and there will be more or less ascites, the physical signs being practically those of a chronic tubercular peritonitis. In all cases, whether primary or secondary, the clinical picture, though varying enormously, shows a rapid and extreme degree of emaciation, often without any fever, and with which is associated the peculiar cancerous cachexia. There may or may not be pain and tenderness, but as a rule the neoplasm is prone to excite, as in the case of the tubercle bacillus, a marked degree of inflammatory change in the peritoneal membrane, which may result in adhesions and extensive matting together of bowels and viscera. Since, then, the symptoms and signs are those of a chronic tubercular peritonitis in most cases, it is needless to recapitulate their endless variations, the main points in the differential diagnosis being that—in cancer: (1) The development of emaciation is rapid and progressive, whilst in the tubercular affection it is marked by periods of considerable improvement, in which the body-weight of the patient may notably increase and the anæmia disappear. (2) Peritoneal cancer, especially when secondary, as it usually is, occurs about or after the age of forty, whilst tubercle is almost always earlier. The writer has, however, witnessed the diffuse endothelial type in several cases in young subjects under thirty which was, except in one instance, primary. (3) In the one case cancer may be found in other organs, as the breast and uterus, and in the other tubercle in the chest or elsewhere will settle the diagnosis. (4) The negative effect of small tuberculin-injections sometimes affords valuable evidence in favour of the malignant affection. (5) A marked family-

history of cancer is of more value from a diagnostic point of view in malignant cases than a family-history of tubercle in tubercular cases. (6) The diagnosis is easy when the previous symptoms indicate a primary cancer of the stomach, liver, ovary, or uterus. (7) Secondary nodules of cancer are often felt in the abdominal wall, especially in the vicinity of the umbilicus; whilst inflammatory redness and general thickening occur not very rarely in tubercular peritonitis, with or without an umbilical fistula. The writer has confidently diagnosed the diffuse malignant affection when the only sign was the retraction of the navel, which was surrounded by a steel-like ring under the skin as the umbilicus was pulled in by the shortening of the fold of peritoneum containing the foetal remnants of the urachus and hypogastric vessels. (8) The ascitic fluid is more frequently hæmorrhagic in cancer than in tubercle; but too much importance should not be attached to this sign. (9) Of much more value is the discovery of nests or groups of multinuclear cells in the ascitic fluid in cancer and of the tubercle bacillus in tubercular peritonitis.

SARCOMA OF THE PERITONEUM

Etiology and Pathology.—This is generally of retroperitoneal origin, and many authorities regard the majority of cases of malignant peritonitis as examples of round or spindle celled neoplasms; they may also occur primarily in the omentum or mesentery. They show little tendency to infiltrate the abdominal viscera; they displace these in various ways as innocent growths do, but they are much more liable to cause pressure on the portal vessels, producing œdema of the legs, and ascites, with distension of the superficial veins. Unless when they occur in the omentum, they are seldom movable, and they tend to become cystic as their development progresses. Their diagnosis from primary or secondary carcinomatous growths is often impossible, and, moreover, it is of no great importance, since both are uniformly fatal and beyond the reach of operative interference. The differentiation of sarcoma from *scybulous impactions* can often be only made after the persevering use of the enema-tube.

Lipomatous neoplasms, fibromata, nævoid growths or angeiomata, and dermoid and hydatid cysts are occasionally met with in the peritoneum; their rarity gives them a museum-interest only.

Simple cysts are not rarely met with; they are probably of inflammatory origin, or they may be caused by dilatations of lymphatic vessels, since some of them contain chyle. Under 'Ascites' the condition sometimes called chylous peritonitis is described. Distension or ascites of the *lesser*

peritoneal sac may occur from some inflammatory action set up in the neighbourhood of the foramen of Winslow, obliterating the opening into the general peritoneal cavity. This may occur as the result of tubercle or cancer or of the peritonitis produced by septic organisms.

PERITONITIS, ACUTE

Inflammation of the peritoneal membrane, acute or chronic, very seldom, if ever, occurs as a primary disease.

Etiology.—The most common cause of acute general peritonitis is the rupture of a gastric, duodenal, or intestinal ulcer, or an appendicular, hepatic, renal, splenic, or pelvic abscess, or an abscess originating in the gall-bladder. In all these cases the type of peritonitis is usually styled the *Perforative*, and in every instance the inflammation is the result of the introduction into the peritoneal cavity of micro-organisms, usually of the pus-forming class.

In inflammation of the different organs within the abdomen the same result follows without perforation by means of the direct extension of the inflammatory process from the organ to its peritoneal coating. That this extension is effected by the spread of similar organisms cannot be doubted, and in considering the etiological factors at work the main prominence must be given to sepsis, which is the constant feature in every case of peritonitis, as will be detailed in the description of the pathological anatomy.

Obstruction of the bowel and hernia cause general peritonitis in the same way by permitting pathogenic organisms to find their way into the peritoneal sac. The so-called '*idiopathic*' type of peritonitis is supposed to be often of rheumatic origin, but as yet all the organisms which can set up acute inflammation in the peritoneal membrane have not been isolated or identified. In the meantime it is safe to infer that most, if not all, cases grouped as idiopathic are but instances of an infection probably secondary to the same process started in some other part of the body.

In this way the peritonitis following inflammation of the biliary passages and any of the organs in the pelvis, especially in the female pelvis, is to be explained. Pneumonia and empyema are more frequently the result than the cause of acute peritonitis, but undoubtedly the organisms which produce these diseases may cause a secondary peritonitis even when there is but little evidence of their primary attack on the pleura. It is probable that in rare cases the microbes may reach the peritoneum through the blood-stream and excite inflammation, though even in these instances some local lesion in the skin, the lungs, pleura, bowel, or genito-

urinary passages may have escaped detection. This latter is not unlikely to be the case in gonorrhœal peritonitis in the female when the local lesion in the vagina or Fallopian tubes is so slight as to escape suspicion; and in the peritonitis associated sometimes with erysipelas and septic wounds in distant parts of the body the organisms are probably not always conveyed in the blood-stream. The terrible mortality following the earlier operations before the introduction of aseptic methods for ovarian disease, strangulated hernias, &c., has demonstrated the liability of the direct introduction of various germs to excite a fatal inflammation of the membrane. The extensive and prolonged abdominal operations of the present day have also demonstrated the enormous amount of injury which may be inflicted upon the membrane without any inflammatory action supervening, as long as due precautions are rigidly undertaken for the exclusion of all microbes. Accidental abdominal wounds are, of course, much more liable, for the above reasons, to be followed by peritonitis than are those inflicted by the surgeon. Bright's disease, gout, and diabetes in a small percentage of cases are associated with acute peritonitis, in which case the latter is a terminal event; the occurrence of peritonitis under these conditions does not prove that the inflammation is not of microbic nature. The theory is very probably correct that this variety is of the same type as all the others—viz. that it is due to micro-organisms which are constantly finding an entrance to the blood or to the peritoneal sac, but whose virility is continually being destroyed by the process of phagocytosis provided for by nature in the peritoneal membrane and elsewhere. The renal lesion, through its ulterior influences upon the blood and phagocytes, acts by diminishing the natural resistance of the serous membrane. This is the probable explanation of the peritonitis which follows not rarely upon chills caused by exposure of the body or of the abdominal wall to direct cold. This diminished resistance is now a well-recognised etiological factor in the production of such unquestionably microbic diseases as pleuritis, empyema, pneumonia, and probably acute rheumatism. An epidemic form of the disease has been reported by several observers, but the specific organism has not been demonstrated.

Sex acts as an etiological factor, inasmuch as it increases the danger of infection from the vagina, uterus, and its appendages; and the majority of perforating gastric ulcers occur in women. Whilst peritonitis occurs at all ages, its causes vary with each decade, being chiefly due in the first twenty years of life to intussusception, acute strangulation, and tubercle; at a somewhat later period appendicular inflammation and gastric ulcer operate most frequently. The child-bearing decades supply the majority of the pelvic cases, and strangulated

hernia operates as a frequent cause in strong muscular males, usually about the period of early middle-life.

Tubercle as a cause of peritonitis will be referred to later on under a separate heading; though usually productive of a chronic type of inflammation, occasionally its deposition is swiftly followed by a very acute adhesive inflammation.

Morbid Anatomy and Bacteriology.—The changes are practically identical with those observed in inflammation affecting other serous membranes, but we are more familiar with them in their earliest stage in peritonitis owing to the frequency with which they are rendered visible in laparotomies. The membrane is congested in patches, loses its polished shining appearance, and conveys a distinctly sticky feel when handled. This is seen at first in its most intense degree between the coils of the distended bowel, or where the tension of the gas in the intestine forces the blood into that part of the tube subjected to the least direct pressure from without.

Serous effusion rapidly occurs: sometimes from the first it is purulent in nature; an exudation of yellowish-white fibrinous lymph is found covering over the distended coils of bowel and filling in the interstices between them. Sometimes this exudate shows early stratification and may be peeled off in layers; at a later stage, if life be prolonged, it becomes organised into granulation-tissue, causing tough and dense adhesions. The amount of fluid is seldom very great, and it is usually diffused generally over the entire abdomen owing to the pressure of the distended coils of intestine. Amber-coloured at first, it gradually deepens in hue, becomes green, and in the perforative cases rapidly changes from translucency to various degrees of opacity, and finally may become obviously purulent, and fetid in odour. In mild cases the effused fluid may remain transparent throughout, and at a later stage it may be entirely absorbed along with the fibrinous exudation.

The changes vary much in extent and in degree of intensity according to the nature of the primary lesion, being sometimes quite localised and at others involving the entire visceral and parietal layers of the sac. When strictly localised, the inflammatory process may be shut off completely from the general abdominal cavity, in which case the circumscribed products of inflammation may be converted into an abscess-cavity situated between two coils of bowel, but more often in a pelvic or sub-diaphragmatic pouch.

Where the inflammation has been the result of a perforation of the stomach or other part of the intestinal tube, the gaseous and liquid or semi-solid contents escape into the general abdominal cavity, unless the opening has been of the pin-hole variety, in which case the contents may be encysted or shut off by inflammatory exudation and adhesions.

In chronic cases all sorts of bands, ribbons, or cords may be found as the result of the stretching of adhesions by the vermicular contractions of the bowel.

Of the various organisms found in the lymph and fluid effusion upon bacteriological examination the most common is the bacillus coli. Experimental evidence has demonstrated that this organism, always present in a harmless condition in the human intestine, is prone to undergo morphological changes and to develop an extraordinary degree of virulence when any injury has occurred to the intestinal walls. It is able to pass readily from the mucous to the serous coating of the bowel in strangulations such as occur in hernias and in diseased conditions like appendicitis, diarrhœa, &c., in which no abrasion of surface is present. It is nearly always present in the peritoneal inflammations caused by disease of the gall-bladder and bile-ducts. In perforative peritonitis it is almost constantly present in association with streptococci, and less frequently with staphylococci, pneumococci, bacillus proteus, and many other pyogenic organisms.

In the so-called idiopathic cases, streptococcus pyogenes, staphylococcus aureus, micrococcus lanceolatus, and others have been recognised in addition to the bacillus coli. That some of these cases even from the strict bacteriological point of view are to be regarded as secondary is rendered probable by a case carefully investigated recently by Lorenz. It occurred in a boy, and the diagnosis of the cause of the peritonitis was appendicitis, but this appendage at the laparotomy was seen to be normal. After death the staphylococcus pyogenes aureus was found in abundance in the peritoneal effusion everywhere. A slight excoriation on the little toe had caused a lymphadenitis, which extended to the retroperitoneal glands; and the staphylococci were also found in the affected lymphatics. But for the thoroughness of the investigation which led to the discovery of the insignificant septic wound such a case would have been regarded as typical of a primary or idiopathic inflammation of the peritoneal membrane. The typhoid bacillus and the microbe of influenza have been identified in several cases in the effusion.

J. D. Malcolm still insists upon the 'not uncommon condition of an obvious definite *aseptic* peritonitis, the inflammation being the result of physiological processes set up for the absorption or demarcation of the damaged tissue.'

Dudgeon and Sargent, in their recent 'Erasmus Wilson Lectures,' state that they found in almost every variety of peritoneal lesion an organism of very weak virulence—the *Staphylococcus albus*. This they believe to be the cause of the febrile reaction so constantly found after the effusion of blood into the peritoneal cavity. An identical or similar organism has been detected frequently in *aseptic* wounds by

Walsh. Sir Lauder Brunton believes that the rise of temperature in peritonitis, as in other acute inflammations, may be the result not of microbic action, but of a ferment—plasmase—produced by the injury of the white corpuscles. The question of leucocytosis has been discussed in connection with appendicitis.

Under the term *Polyorromenitis*, Italian physicians describe a form of acute suppurative peritonitis which is found associated with suppurative inflammation in other serous membranes. The combination does not, however, justify the condition in being regarded as a distinct disease. The bacteriological examination of the purulent effusion, which may appear first in the peritoneum and afterwards in the right and subsequently in the left pleura, proves that the condition is the result of a general infection. Staphylococci, pneumococci, streptococci, tubercle and influenza bacilli, and other organisms have been isolated, as in those cases of acute suppurative peritonitis in which the mischief has remained confined to the peritoneal sac throughout. It is worthy of note, however, that, according to the statistics of Picchini, half the cases recover when the pus is evacuated.

Netter states that in autopsies on pneumococcus-pneumonia he was constantly able to demonstrate the presence of the pneumococcus in cover-slip preparations from the peritoneum, though no signs of inflammation were present, and his observations are confirmed by Flexner; so that pneumococcic peritonitis is probably more common than is usually suspected. Matthews states that it is prone to become localised in the pelvis and hypogastrium, forming a large thick-walled abscess in these situations. The exudate is very fibrinous, odourless, and of a greenish-yellow colour, and the tendency is towards recovery as soon as the exudation is removed.

In the peritonitis caused by actinomycosis, involving the liver and cæcum, the ray-fungus has been found in the purulent effusion, which is generally circumscribed or loculated.

Symptoms.—In every case of severe acute peritonitis the clinical picture may be said to be painted in two prominent colours, and at the bedside these should be separately recognised if the physician is to grasp the meaning of the symptomatology of this grave and often puzzling disease: (1) There are local symptoms and signs pointing to the abdomen or lower thoracic region and pelvis; these at a later stage are often entirely overshadowed by (2) the grave constitutional *toxæmia*, the result evidently of the absorption of the toxins manufactured by the micro-organisms, which rapidly multiply upon the extensive sheet of inflamed membrane.

The symptoms are observed in their most typical form in the perforative variety of peritonitis, especially when the inflammation occurs in an individual supposed to be in

ordinary good health, though suffering from, say, a latent gastric ulcer.

The onset is sudden or abrupt, setting in sometimes with symptoms of shock, and not unusually accompanied by a rigor or severe chilly sensation, and always with intense abdominal pain. The pain cannot usually be located in any particular spot on its first abrupt onset. It is felt diffused over the entire abdomen, though the patient is usually able to discriminate its localisation to the extent of expressing that its headquarters are situated somewhere in the neighbourhood of the umbilicus. In character the pain is as varied as is the natural degree of individual sensitiveness, but it is always more or less agonising. Unlike the sudden seizure of colic, the pain is invariably associated with very great tenderness of the abdominal skin, amounting to a highly marked degree of hyperæsthesia. The patient shrinks from the lightest touch and cannot tolerate the pressure caused by the weight of his bed-clothing. Change of posture and all muscular movements aggravate the intensity of the suffering, whilst absolute rest may give slight relief. After some hours the patient is often able to definitely locate the site of his pain, which varies, of course, with the situation and nature of the primary lesion. In stomach-perforation it may be felt behind close to the spine, or in front just below the great curvature and to the left of the umbilicus. In appendix-trouble it may be exactly referred to the inflamed spot, as verified by a subsequent incision; often, however, in many varied conditions, it is finally localised about or below the umbilicus.

One feature of the pain should be watched for—when present it helps to aid the diagnosis of the origin of the peritonitis in an acute strangulation: this is its paroxysmal nature, the seizures coming on with a degree of regularity strongly suggestive of the intense effort made by the bowel to overcome some formidable obstruction. This is never seen in ordinary perforative peritonitis.

The hypersensitive abdominal parietes are rigidly contracted and as unyielding as a plaster-cast. The patient lies upon his back, with his knees elevated and the soles of his feet flat upon the mattress on which he lies with his shoulders somewhat raised and often with his arms elevated above or behind his head.

Very rapidly the bowels become distended with flatus, and sometimes the sac fills up with gas from the bowel or stomach. This tympanitis of the belly may be marked inside twenty-four hours after the onset; it is greatest in acute peritonitis following acute obstruction, and may be absent throughout in septic cases. Though the abdomen is tense from the gas which distends it at this stage, the rigidity

of its walls may entirely pass off. The breathing is of the rapid superficial or costal type owing to the inhibition caused by the inflamed diaphragm and the abdominal tympany.

Nausea sets in very early and vomiting soon follows ; this, after the evacuation of the contents of the stomach, consists of bile-stained fluid, which is incessantly brought up with much distress and aggravation of all the symptoms. Usually foul eructations are associated with it, and as the evacuated matter changes to a greenish-brown or black the odour becomes distinctly feculent and finally stercoraceous. Vomiting is often entirely absent in cases of perforation of the stomach. Constipation is the rule in peritonitis from whatever cause, save in typhoid-ulceration and in dysentery. Thirst is intense ; the tongue, at first foul and coated, becomes dry and brown like sole-leather. Fever is usually present and sometimes runs high ; in the initial stage of shock the temperature is often sub-normal. At a later stage the absence of fever is a not uncommon event, but its presence or absence affords no means of gauging the gravity of the situation. It may be even unnoticeable or slight in the most rapidly fatal forms of the disease.

The pulse changes rapidly, being quick, small, and thready or wiry, easily obliterated, and by the end of forty-eight hours it may be uncountable ; before this terminal event it may register 160. The condition of the pulse is of vital importance in prognosis, being sometimes excessively rapid and small when the other symptoms may not suggest a serious issue.

As the development of the above symptoms becomes complete the clinical picture gradually or speedily changes into one of profound septic poisoning in all cases which do not recover.

The patient presents the aspect of one in a grave state of exhaustion : his features are pinched, his nose being sharpened, his cheeks pallid, leaden, or of ashy hue ; his eyes are sunken, and the expression is one of anxiety and extreme restlessness. Perspiration appears upon the wrinkled brow and spreads over the gaunt hollow cheeks, and bathes with clammy moisture the cold and often livid hands and feet. He gradually sinks from asthenia, with his intellect unclouded, apparently often free from fever, though the thermometer in the rectum may register 106° immediately before death, whilst the surface of the body feels cold and the extremities are cyanosed, and all traces of pain and abdominal tenderness may have passed off.

There is sometimes evidence of the collection of a considerable amount of fluid on gentle percussion of the abdomen, but owing to great intestinal tension and the abundant adhesions it does not speedily gravitate towards the lowest level on changes of posture. Dulness is first observed in the

loins ; the signs vary when gas has been freely admitted to the general peritoneal sac, as the air always occupies the highest level as soon as the posture alters. Much importance has been attached to the alterations in the splenic and hepatic dulness : the former usually disappears entirely, but the liver-dulness, though diminished, may generally be demonstrated to be present, unless the diaphragm is greatly displaced upwards. Obliteration of the entire hepatic dulness is usually regarded as distinctive of perforative peritonitis ; but this is not always the case, unless there be much free gas in the abdominal cavity. As stated under 'Gastric Ulcer,' a very valuable sign should always be looked for in cases of supposed perforative peritonitis from ulcer of the stomach : it is the absence of the true gastric note on careful percussion, which may be taken as indicative of the escape of gas from the organ. The heart is generally much elevated and the lungs may be considerably compressed in their lower portions. The course of the disease varies widely in different cases : perforation of the stomach or of a dilated colon may in rare cases prove fatal from shock before the development of the inflammation. As a rule, such cases terminate fatally inside about forty-eight hours, unless relieved by operation. Strangulation and hernias set up an acute peritonitis which runs a somewhat slower course, the fatal issue being often postponed for four or six days or more. The duration of the illness is liable to be lengthened when adhesions form early and shut off the inflamed area from the general peritoneal sac. In these latter cases, as typified by disease of the appendix, pus may form in the isolated pouch of serous membrane, which, on bursting into the general abdominal cavity at a later stage, will produce an intensely rapid form of acute general peritonitis.

Diagnosis.—The chief points have been already emphasised in the description of the various individual symptoms which blend to form the clinical picture. The main effort of the physician should be to satisfy himself that the peritoneal membrane is in a state of acute inflammation, and not to waste time in waiting for the development of symptoms which will enable him to diagnose the cause of the peritonitis. To do so is often to sacrifice life, since in many cases the only hope of relief lies in speedy resort to operative interference. For the same reason morphia, indicated by the agonising pain, must be sparingly used, so that narcotism shall not on the one hand mask the symptoms, or upon the other lead the surgeon to the conclusion that a hopeless septic toxæmia has already set in. The history of the patient's previous illnesses will in many cases throw considerable light upon the case as soon as the physician is called in for the graver ailment. Thus the history of the symptoms pointing to gastric ulcer,

especially if hæmatemesis has ever occurred and the patient is a young female, is almost conclusive. The knowledge of previous attacks of a recurring appendicitis should direct attention immediately to the right iliac fossa. Typhoid perforation may be reasonably anticipated in severe cases where the constitutional and local symptoms point to deep ulceration of the intestinal mucous membrane. Remembrance of the etiological factors mentioned in connection with age and sex often gives a clue to the cause of the attack.

Colic should seldom be mistaken for peritonitis, for, as already pointed out, in this condition there is not only absence of hyperæsthesia, but deep and firm pressure over the retracted abdomen is often instinctively employed by the patient to procure relief; vomiting, tympanitis, and all the constitutional signs, as fever, weakness of pulse, and toxæmia, are absent; whilst signs of lead-poisoning may be present.

Hysterical pain in the abdomen may simulate the onset of peritonitis, but the absence of grave constitutional signs and the presence of some of the characteristic neurotic symptoms are certain to betray themselves if carefully looked for.

Acute intestinal obstruction, whether caused by bands, volvulus, intussusception, gall-stones, hernia, &c., usually begins with symptoms resembling acute general peritonitis; at a later stage the clinical picture may be absolutely identical, since the tendency is for the terminal phenomena always to eventuate in peritonitis. The differential diagnosis is of importance in so far that the physician should be able to recognise the inherent symptoms of the obstruction without waiting for the advent of general peritonitis to confirm his diagnosis, in which latter case the prospect of operative interference may be hopeless. The absence of the early general abdominal tenderness and rigidity, the more marked paroxysmal nature of the pain, the speedier advent of distension, the greater predominance of the vomiting, and the liability of the latter to become rapidly fæcal, are points of much diagnostic importance in acute obstruction when considered in association with the absence of fever and of interference with the movements of the diaphragm.

The diagnosis of *localised* peritonitis is comparatively easy in the form most commonly to be met with—viz. that following appendicitis. The local signs, often pointing to the formation of pus and the presence of obscure or obvious fluctuation in the appendicular region, seldom permit of much doubt when considered in association with the history of the onset and progress of the disease.

Sub-phrenic Abscess is rarely the result of a localised peritonitis originating in the liver or gall-bladder. As the abscess-cavity is shut off from the general peritoneal sac, rupture into the pleural cavity may occur, especially on the right side,

setting up pyothorax. The most common form of sub-phrenic abscess is that following the perforation of an ulcer of the stomach, more rarely of the duodenum. The escape of stomach-contents and gas into the isolated sac of peritoneum produces a remarkable condition, which has been described as *sub-phrenic pyopneumothorax*. It may occur on either the right or left side—more frequently on the latter, in which case a considerable space containing air and pus is bounded above by the left half of the dome of the diaphragm, which is pushed high up into the thorax, its lower boundary being formed by the left lobe of the liver and the stomach, which become adherent to the abdominal wall, whilst the peritoneal covering of the abdominal parietes shuts it in anteriorly; laterally it is bounded by the falciform ligament on the one side and the false ribs on the other. The ultimate course taken by the pus will cause considerable variations in the signs. Pyothorax or pyopneumothorax may result, or the sub-phrenic abscess may discharge itself by the mouth after bursting directly into the lung. It is to be remembered that this condition may be caused by other than gastric ulceration; it sometimes follows the rupture of an appendicular abscess, disease of the colon, duodenum, or pancreas. In some cases a simple sub-phrenic abscess, originating in the liver, gall-bladder, or pancreas, may afterwards perforate the stomach, intestine, or even the lung, in which case gas is freely admitted to the isolated sac. In perforation close to the duodenum the cavity forms sometimes to the right of the falciform ligament of the liver and pushes up the diaphragm and depresses the liver considerably.

The symptoms are those of a localised peritonitis following the shock of the perforation; there is severe pain, associated always with dyspnoea and most marked interference with the movement of the diaphragm, but this movement is usually restored at a later stage. After the rapid formation of pus which follows, the constitutional symptoms are those of any acute internal suppuration—rigors, pyrexia, sweating, &c. Unless the cavity contain gas, it is not likely to be mistaken for an ordinary pyopneumothorax; where gas is absent the signs are those of pyothorax, but the very low level of the exudation, the great depression of the liver, and the absence of lateral displacement of the heart are almost characteristic.

Peritonitis, limited to the small sac of the peritoneum through closure of the foramen of Winslow, may result from perforation of the posterior wall of the stomach, duodenum, or colon, or from abscess of the pancreas; it sometimes occurs in chronic tubercular peritonitis.

Torsion of the pedicle of an ovarian tumour causes symptoms closely resembling peritonitis—indeed, Malcolm recently, in his valuable contribution on the physiological processes of peri-

tonitis, regards this condition as typical of an acute *aseptic* inflammation of the peritoneum; the inflammation, however, does not spread, and the diagnosis is easily made when the tumour-history is investigated. *Ruptured extra-uterine foetation* produces peritonitis of a similar apparently aseptic type, whose symptoms may be entirely those of a large peritoneal hæmorrhage, causing shock and collapse from its injury to the abdominal nervous system—a condition described as *Peritonism*.

Prognosis.—This is always grave in acute general peritonitis; the cases which recover are mainly those associated with operative appendicitis or with the inflammation following gastric perforations. The prognosis is largely influenced by the possibility of the removal of the immediate cause by surgical measures, and the hopefulness of the outlook brightens considerably with the promptitude of the interference. Recent years have steadily demonstrated the diminution of mortality in perforative peritonitis, as the physician has learned to value the importance of timely operation. Where operative interference is not indicated, the mortality is therefore obviously very high. It is almost always fatal in aged subjects. In infants the mortality is also very high; in the form which occurs soon after birth, from the infection of the stump of the umbilical cord, recovery seldom occurs.

Though the prognosis in all cases may be said to depend largely upon the primary cause, often without being able to determine this, an accurate forecast of a fatal result may be safely made by an analysis of the symptoms. Where these are distinctly of the *toxæmic* type, when the patient is first seen, the case is certain to end fatally. The pulse is a valuable criterion, and sometimes betrays the gravity of the case when every other sign and symptom may be wanting, as in some forms of so-called *latent peritonitis*, which are prone to become purulent. Extreme restlessness is a grave sign, and so also is hiccough. The sudden disappearance of pain, without improvement in the other symptoms, is of evil omen, and usually indicates a high degree of poisoning, which will be soon followed by collapse.

In pelvic peritonitis the outlook is better as a rule than when the upper zone of the abdomen is the site of the first symptoms.

The acute peritonitis occurring in the late stages of Bright's disease is usually purulent, and always fatal. When peritonitis is associated with erysipelas of some distant part of the cutaneous surface the result is always rapidly fatal.

Treatment.—This subject is a wide one, and has been dealt with in detail in the author's 'Dictionary of Treatment' (4th ed.), pp. 685–696; only the main principles need be here referred to. The first indication is the removal of the primary

exciting cause by operative interference, without waiting till the entire peritoneal sac becomes infected. In spreading peritonitis, such as follows rupture of a gastric or intestinal ulcer, or inflammation of the appendix, immediate opening of the abdominal cavity is demanded as soon as the patient has recovered from the initial shock. In acute general peritonitis, when once the disease has become established, a fatal issue may be confidently expected, and the trend of opinion is distinctly in the direction of a similar course. By a free incision, and the thorough flushing out of the entire peritoneal sac with warm saline solution, life may be sometimes saved in most unpromising cases. Ballance goes so far as to recommend that after a general infection of the sac has occurred, the patient's abdomen having been opened and flushed out, he should be immersed in a bath of saline solution, so that the liberated intestines may be permitted to float in the fluid.

Murphy's recent method of treating acute general peritonitis is the one most generally accepted as the best in all serious cases. It consists in the immediate opening of the abdomen, the quick removal of the origin of the inflammation, and the establishment of drainage, with the minimum possible disturbance of the viscera. In the *diffuse general peritonitis* following appendicitis, for example, after the operation he keeps the patient in a sitting posture at from thirty-five to forty-five degrees, and administers a quart of saline solution per rectum every two hours. He discourages strongly the mopping out, washing, or handling of the peritoneum, and relies upon putting a stop to all pus-absorption by the simple opening of the abdomen, which relieves all pressure and exposes the anaërobic flora to the air. By these means he has, in acute generally diffused peritonitis, treated twenty-nine cases with but one death, which occurred from pneumonia upon the sixth day.

When operative treatment is not indicated or possible, the management of the case resolves itself into the use of such measures as will afford relief to the most urgent symptoms. Rest is essential; pain must be relieved by morphia hypodermically when its intensity is not mitigated by poultices, warm fomentations, ice-bags, or leeching. Unless the case is hopeless from the beginning, the routine administration of large doses of opiates is condemned by all authorities, for the reason already mentioned—that narcotism masks the symptoms and tends towards the deepening of the toxæmia.

Feeding must be carefully attended to; the less material introduced into the stomach the better, the rectum being selected always as the route when this is practicable. Thirst is often the most distressing of all the symptoms, being more difficult to bear than pain in many cases. Small quantities of

ice sucked in the mouth may be permitted, and an occasional flushing of the colon with warm water, followed by a large enema, administered with the view of its being retained, often gives relief. Often strychnine hypodermically affords great relief.

Vomiting may be alleviated by minute doses of morphia, frequently repeated, and occasionally a copious draught of warm water may be administered with the view of its being ejected, so that the stomach may be washed out; but such a measure is clearly contra-indicated in perforation of the organ. Tympanitis may be occasionally relieved by copious watery or turpentine enemata: the passage of the long tube has never been seen by the writer to do any good; perforation of the distended coils of bowel with a small trocar is a last resource, and often useless. Saline purgatives are invaluable in the symptoms closely simulating the advent of peritoneal inflammation which appears often in cases after abdominal operations, but the routine administration of salines in acute peritonitis must be condemned.

PERITONITIS, CHRONIC

Like the acute disease, this is seldom, if ever, primary.

Etiology.—The condition is sometimes the sequel to acute inflammation, especially when the septic infection has been of such a mild degree of virulence that phagocytosis has been able to retard its progress, or adhesions have been able to prevent the implication of the entire sac. In this latter case the isolation of the inflammatory products may be followed, at a much later period, by a chronic adhesive type of inflammation which, starting in the neighbourhood of the original localised peritonitis, may gradually involve a large portion of the serous membrane, ending in the condition usually described as *diffuse adhesive peritonitis*, in which the general sac becomes finally obliterated. A similar result is characteristic of chronic tubercular peritonitis. Much more frequently, however, the adhesive process itself becomes localised; this condition is so very constantly met with in opening the abdominal cavity, long after laparotomy has been performed, that Herman maintains that *local adhesive peritonitis* is an invariable sequel of abdominal section. The condition is very frequently the result of chronic inflammation, originating in the liver and spleen, which, after involving the capsules of these organs in a perihepatitis or perisplenitis, spreads to the surrounding peritoneum and involves the diaphragmatic covering. Often the origin of the changes in these organs is due to granular kidney or to syphilitic and gouty poisons, but frequently it is the result of long-standing

venous blood-stasis. Pleurisy and pericarditis may be the starting-point in some cases, and uterine or other pelvic inflammations very frequently eventuate in a localised chronic peritonitis. The condition is constantly met with in operations undertaken for the relief of gall-stones and gastric ulcerations, in which the organs in the upper part of the abdomen are found matted together or fused into an apparently conglomerate mass by the gradual extension of the original inflammatory action.

This diffused adhesive inflammation in these latter-mentioned cases is often unassociated with clinical symptoms of any kind save those produced by the primary disease, and the history generally does not warrant the assumption that at any period there has ever been evidence of an acute attack of peritonitis, the process being from the first of a chronic nature.

Under the term *Proliferative peritonitis* Osler describes the variety of chronic peritonitis whose main feature consists in great thickening of the peritoneum, with but scanty adhesions; the omentum is rolled up and the mesentery shortened, though the intestines are free. This condition, met with often in the subjects of chronic alcoholism suffering from hepatic cirrhosis, differs only in degree from the varieties of adhesive peritonitis caused by chronic gastro-intestinal lesions, the sites of the primary mischief in these latter cases accounting for the gluing together of the intestinal coils, which may escape when the mischief originates in the capsule of the liver, or seems to start in the peritoneal layers which pass between the different viscera. Toxæmia is supposed to be the cause of chronic peritonitis in many cases; thus Hale White regards the peritoneal changes associated with perihepatitis and perisplenitis as the result of an inflammation caused by alcohol in the peritoneum and not in the underlying organ. Gout, lead, Bright's disease, and arterio-sclerosis are supposed to act in a similar manner,

Ascites of long standing, from whatever cause, is believed to excite a chronic inflammation of the peritoneum; doubtless, however, in many cases its origin may be explained by the introduction of organisms in some tapping operation, and this may be the explanation of the chronic peritonitis and adhesions which were so frequently met with when tapping of large ovarian cysts was the rule before the practice of ovariectomy.

Trauma acts in the same way, but occasionally chronic inflammation follows contusions without skin-wounds, in which case the peritonitis, in the writer's experience, is liable to turn out to be of tubercular nature. In the absence of any of the above etiological factors the disease is regarded as of *idiopathic* type, when there is no evidence of malignant

growth, which is also often a cause of chronic peritonitis. Chills and rheumatism are supposed to be the explanation of these so-called idiopathic cases, but there is no scientific data to support the theory. Possibly the discovery of the presence of the mild *Staphylococcus albus*, which has been demonstrated by Dudgeon and Sargent in cases where blood has been effused into the peritoneal sac, will clear up the mystery which surrounds this group of cases.

Pathological Anatomy.—The changes met with are those seen in the chronic inflammations of other serous membranes. The peritoneum is opaque and thickened, and usually deeply pigmented in patches. The newly developed fibrous tissue shows its inherent tendency to contract, as seen in the shortening and kinking of the mesenteric attachments and the gluing together of the coils of bowel by dense adhesions, which connect them sometimes firmly to the spine behind or the abdominal wall in front. The shortened great omentum is rolled upon itself, and forms a thick band stretching across the upper part of the abdomen. The solid viscera have their capsules more or less thickened, and are often adherent to the diaphragm, abdominal wall, and to different portions of the gastro-intestinal tube. Bands of fibrous tissue may be found stretching like cords or ribbons from one viscus or coil of bowel to another, and, as in the shorter adhesions, large vessels may be found in these which develop in the newly formed fibrous tissue. The degree of adhesion varies greatly; sometimes the entire sac is obliterated, at other times it may be found divided into several more or less distinct compartments. Often small tuberculated nodules of fibrous tissue may be seen scattered over the peritoneum, which are liable to be mistaken for tubercle, cancer, or disseminated sarcoma. Serous effusion is commonly present, but its amount varies within as wide limits as does the extent of the adhesions; often they are in inverse ratio, a copious fluid exudation, existing with sparse adhesions and an entire matting together of the organs, being present without any fluid; with the entire obliteration of the cavity, obviously no effusion can take place. The fluid is usually of the character of ascitic effusion, the fibrin having disappeared; it may, however, be purulent or hæmorrhagic. The spleen, liver, and kidney, under their thickened capsules, not unfrequently exhibit marked cirrhotic changes, and the bowel itself is often shortened in length and diminished in calibre.

Symptoms.—These may be entirely absent, the condition being only recognisable by the signs which the effusion produces. In other cases there may be no signs, the state of matters being then only to be inferred by the symptoms caused by the adhesions. All sorts of combinations of these signs and symptoms may be met with.

A feeling of distension is common, and sometimes pain and tenderness are complained of over a considerable area of the abdomen. The pain being usually caused by the dragging upon bands of adhesions, situated between a fixed and movable point, it is usually made worse by movements of the body or by the distension of the stomach or bowel by food or gas, or it may only be experienced when the patient is lying in a particular position or attitude. Constipation is the rule, and sometimes this amounts to obstruction. Vomiting occasionally occurs, the appetite is poor, and very commonly emaciation is marked. Fever is not present, except in occasional paroxysms caused by some sub-acute attack of inflammation, probably caused by a temporary constriction of some knuckle of bowel, in which case there will be rhythmical colicky pains coming on some time after eating. In a recent severe case under the writer's observation, the symptoms were considered to be due to a typical duodenal ulceration, for which the patient was sent into hospital; the laparotomy revealed nothing but extensive gastric adhesions following an old operation for a catarrhal appendicitis, and the symptoms entirely disappeared after a gastro-enterostomy was performed.

The signs are very variable. Sometimes a friction-fremitus may be felt; when the amount of effusion is great the abdomen presents the general features of ascites, but even then the enlargement is rarely symmetrical. Owing to the frequency with which the fluid is encysted, the sense of fluctuation is often obscure, or it may be detected easily in two or more different isolated regions. Very often doughy tumours may be palpated, almost identical with those felt in a multilocular ovarian cyst when the main reservoir has been drained by tapping. The dulness does not usually vary with changes of position, as in ascites, even when much fluid is present.

In the absence of fluid the retracted abdomen may yield evidence, on palpation, of a large, somewhat uneven, sausage-shaped mass lying across the lower epigastric region, where the rolled-up great omentum is situated. The writer has been sometimes able to detect the condition, on simple inspection, by the marked retraction of the tissues around the umbilicus, caused by the shortening of the fold of peritoneum, the remnant of that which contained the urachus and obliterated hypogastric vessels. This becomes more evident as the abdomen gets distended by fluid, when a firm steel-like ring of narrow fibrous tissue may be felt by the finger, surrounding the navel, a sign best marked in association with the peritonitis following a diffuse sarcomatous infiltration. Sometimes much may be learned by watching for a time the peristaltic movements noticeable through the thin abdominal wall.

Diagnosis.—The points in the differential diagnosis of *ascites* have been already emphasised in the description of

the signs, but these should not be relied upon unless when taken in conjunction with the evidence obtainable from a study of the patient's history, especially as regards his drinking-habits. The exclusion of an *ovarian cyst* can only be arrived at after very careful palpation and percussion and a scrutiny of the history of the case, pain being rarely present in this latter condition, which is, moreover, very seldom associated with emaciation and intestinal irregularities.

Tubercle, like *cancer*, is excluded always with great difficulty, and only after a very careful scrutiny of the history and progress of the disease, the main points being, in both these conditions, the degree of extreme wasting present and the detection of evidence of these diseases in the lungs, bones, lymphatic glands, or elsewhere. In tubercular peritonitis there may be marked improvement, followed by more or less acute exacerbations, as diarrhoea and constipation alternate, but the steadily progressive nature of the malignant affection as a rule soon declares itself.

Prognosis.—In all forms of chronic peritonitis the outlook is decidedly unfavourable. In the suppurative cases it is always fatal, unless the inflammatory condition is localised, and is such as can be dealt with by surgical methods. The improved operations by which the stomach can be made to open directly into the intestines, by the various modifications of gastro-enterostomy, afford a hope that much may be achieved in any case by this method of relieving the obstruction caused by adhesions high up in the intestines. Constipation, vomiting, pain, and emaciation are, like other symptoms, to be met by careful and improved methods of feeding, together with the administration of drugs suitable for the relief of each symptom. Sometimes the steady employment of counter-irritants, in the form of flying blisters, may aid in the absorption of localised effused fluid, without resorting to tapping. So-called absorbent drugs, as iodides and mercurials, are valueless for the removal of adhesions, unless in syphilitic capsulitis. When the symptoms clearly indicate interference with the function of the bowel or stomach, an exploratory incision should always be recommended. Often the division of bands or ribbons and the separation of matted coils of bowel are feasible and successful.

PERITONITIS, TUBERCULAR

This, like the other forms of peritonitis, is rarely a primary affection. It is an inflammation of the peritoneal sac, acute or chronic, localised or general, caused by the bacillus of tubercle.

Etiology and Pathology.—These have been already dealt with under ‘Mesenteric Gland Disease,’ in association with which the peritoneal inflammation is so often met, and they differ in no way from the etiological factors which determine the advent of tubercular disease in other regions. The disease occurs at all ages, but the great majority of cases occur between the fifteenth and thirty-fifth year; and it is in the writer’s experience much more frequent in females than in males, as stated by Osler, though the opposite opinion is held by many observers. The bacillus may find its way directly from the intestinal mucous surface, which it has reached through tubercular food, especially when uncooked milk is an item in the diet. The entrance may be effected through a tubercular ulcerative process having been set up in the mucous membrane; but Martin has shown that the bacilli pass through to the serous surface without any abrasion or apparent breach of continuity in the mucous membrane. Under normal circumstances probably the gastric and intestinal secretions are sufficiently powerful to destroy the germs; but when the bacilli are taken into the stomach in great amounts, as when a phthisical patient swallows his sputum, infection of the abdomen is liable to occur. The sac is usually invaded by a secondary infection from the mesenteric or retroperitoneal lymphatic glands, which are primarily involved in most cases.

Tubercle in the thoracic glands and in the lung or pleura may infect the peritoneum through the lymphatics or general blood-stream. The Fallopian tubes in the female and the testicle in the male are the source of the bacilli in many cases, by direct transmission, and this may occur also in some instances of spinal caries.

The presence of tubercle in any part of the body, as in the cervical glands and bones, may cause a rapid or sudden infection of the entire sac, in which case the peritoneal tubercle will probably be but a part of an acute general miliary tuberculosis. In this latter case all the serous membranes may be implicated—*i.e.* the peritonitis will be associated with pleural, pericardial, and meningeal inflammation, constituting a variety of the polyorromenitis or polyoserositis, mentioned upon a previous page (1265), though the exudation be not necessarily purulent.

From the frequency with which abdominal tuberculosis is found associated with carious teeth, especially when a large number of hollow cavities are seen in the mouth, the writer is satisfied that the bacilli multiply in these and are absorbed by the tonsil, thus escaping the destructive action of the gastric juice.

As tuberculosis of the spinal column and testicle frequently follows a blow or contusion, without breach of surface, owing

to the diminished resistance of the tissues to the small amounts of the omnipresent bacilli constantly finding their way into the blood-stream, so tubercular peritonitis often follows injuries to the abdomen and peritoneum. This is the probable explanation of the tubercular peritonitis found in association with cirrhosis of the liver and old hernias.

Morbid Anatomy.—When the peritoneal surface is invaded in an acute miliary tuberculosis, the innumerable, minute, whitish, translucent granulations are scattered over the entire membrane, often occurring most thickly along the under-surface of the diaphragm and over the omentum, which may feel sticky or as if studded over with fine sand. In some places these minute grains coalesce, forming small nodules which, when examined, are found to be raised above the surface and surrounded by a small vascular area. Some lymph is often present, and usually more or less serous effusion; rarely is the liquid purulent from some mixed infection.

Typical tubercular peritonitis is, however, of chronic nature, and more or less fibroid proliferation and caseation are observed in the grey granulations, which are, in advanced cases, seen in the act of breaking down by necrosis. The fibroid changes often predominate, causing extensive adhesions, matting together coils of bowel, omentum, and viscera as in the diffuse proliferative chronic peritonitis of non-tubercular origin, shortening the mesenteries and causing a rolling up of the great omentum, which is much thickened and often infiltrated by caseous masses. With this fibrosis there is often no fluid; the bacilli may be sparse and only to be detected after prolonged investigation, the pathological picture closely simulating that of the syphilitic fibroid degenerative type. Once the writer met with an infecting sarcomatous infiltration of the peritoneum, which he considered to be tubercular, and which was pronounced to be a typical tubercular peritonitis by the experienced surgeon who conducted the autopsy, though histological examination afterwards revealed its sarcomatous nature.

Where caseation predominates, large masses of cheesy material are found scattered throughout the adhesions; the lymphatic glands everywhere in the abdomen are enlarged, and as these undergo softening, necrosis, or ulceration, the intestines are often perforated at several points of contact, where the bowel, glands, and mesentery are matted together. In this way the pathological changes may be wrongly interpreted as the extension outwards of an ulcerous condition from the mucous surface, when its progress has really been from the serous surface inwards; for it must always be borne in mind that the most extensive tubercular peritonitis is commonly found independent of primary intestinal ulceration. Under the above conditions abscesses are liable to

form in loculi between the perforated intestines, and these, as also the rupture of caseating necrosed glands, may precipitate an acute attack of general purulent peritonitis.

There is much pigmentation of the peritoneal membrane, either generally diffused or localised, in dark areas of dense colour, and occasionally calcareous deposits are met with, especially in the mesenteric glands. The amount of serous fluid varies greatly; very occasionally only is it so extensive as in ascites, more commonly it is encysted, or it may be entirely absent, and all degrees from transparent clearness to hæmorrhagic opacity, and even typical purulent fluid, may be observed. When pus is present it may make its way along the obliterated urachus and perforate the umbilicus.

The tubercular bacillus is only found with great difficulty in the ascitic fluid, and often its presence cannot be demonstrated by either the microscope or guinea-pig inoculation.

Symptoms.—In the acute miliary form the symptoms are usually entirely overshadowed by the fever and constitutional disturbance caused by the primary disease or the virulence of the general infection. This variety may therefore be said to be of little clinical importance, save that sometimes it is liable to be mistaken for typhoid fever, and at others it may pass into the typical chronic stage after a period of variable latency. The writer has once observed acute tubercular peritonitis set in with startling abruptness by a large discharge of blood and mucus from the bowel, associated with acute general abdominal pain and high fever, followed afterwards by symptoms of obstruction and much tympanitis. In these cases, though the thermometer may register a high degree of pyrexia, it is of the usual hectic type, with abrupt falls, and often its highest record is observed in the morning hours preceding copious perspiration.

In the ordinary chronic form of tubercular peritonitis the symptoms vary greatly in different cases, and, as in the non-tubercular varieties, the disease, even when extensive, may remain entirely latent—a fact which has been sometimes demonstrated or discovered by the surgeon when operating for the radical cure of an old hernia in an apparently healthy individual, or in the removal of a simple ovarian cyst.

The onset is insidious, as in the non-tubercular chronic form, but the wasting and malaise are more marked often before vague colicky pains, tenderness, and a dragging sensation in the abdomen have been experienced. The abdomen, even at this early stage, may be found distended and tympanitic; a careful routine registration of the temperature will reveal slight fever at irregular periods, often separated by a sub-normal range for several days. The appetite and digestion are disturbed, the tongue is small, red, and irritable, and vomiting at some period of the day is commonly present.

The bowels are constipated at first, but at a later stage one of the most characteristic features of the disease is often present. This consists in spells of obstinate constipation amounting almost to obstruction of the bowel, and these alternate with smart painful attacks of diarrhœa, characterised by foul-smelling liquid motions, in which blood and mucus are sometimes present.

The degree of wasting and the other constitutional symptoms will obviously depend much upon the progress or stage of the primary lesions in the lung, pleura, or elsewhere; but even when these are easily recognisable the abdominal symptoms may be seen partially to clear up for a time, the remissions and intermissions being most variable. Ultimately the physical signs develop in the abdomen; and these also exhibit the greatest diversity and variability in the clinical picture according to the extent and situation of adhesions and the degree of fibrosis or caseation characterising the progress of the tubercular lesions.

In the *chronic fibroid variety* of tubercular peritonitis there may be no tympanitis or meteorism, the abdomen remaining throughout retracted or boat-shaped owing to the shortening of the mesenteric folds, and all evidence of ascitic fluid may be absent. The sense of resistance to pressure will, however, be found to vary considerably over different parts of the parietes, and distended knuckles of bowel may be palpated and percussed in little areas throughout the generally resistant and sometimes doughy mass, which is occasionally found to be devoid of all tenderness. Occasionally the glandular and other tumours about to be described may be obscurely felt. Often a general pigmentation or bronzing of the skin may be observed.

In the ordinary *caseating* and *ulcerative* type there is usually marked distension of the abdomen, either from loss of bowel-tone or from the temporary obstruction caused by adhesions, and the distension is often increased by the presence of fluid. The fluid may be so abundant as to simulate ascites, but usually it is moderate in amount and often encysted, and, as in the ordinary non-tubercular chronic peritonitis, it does not apparently gravitate to the lowest level on changing the patient's posture, owing to the adhesions which prevent the bowels floating upwards. Frequently the presence of fluid cannot be demonstrated when present. As already stated, the fluid upon withdrawal may be clear, bloody, or purulent, and often is devoid of bacilli, so that its characteristics are of little value in the diagnosis; but a very important feature is its variation from week to week, and in this respect it resembles the tympanitis, which is also prone to change from time to time within wide limits, owing to the amount of

obstruction presented to the onward passage of the intestinal gaseous and fluid contents.

The most valuable signs are those obtained by palpation ; unless when the ascitic and tympanitic distension is great, a careful examination will nearly always reveal tumours. These present very diverse characters according to their site and nature. More so than in any other abdominal condition, save in floating kidney, they are prone to vary to the sense of touch from day to day or week to week, because of the fluctuating nature of the effusion and of the meteorism. The most characteristic of these tumour-formations is that caused by the rolling up of the great omentum, which in its folds may contain large caseating masses and even cystic accumulations of fluid. Like a small bolster this irregularly nodulated mass usually lies across the upper zone of the abdomen below the transverse colon and about the level of the navel, but it may be dragged down by adhesions into the right iliac or other region. Occasionally a sharply circumscribed tympanitic note may be produced by *very gentle* percussion when some knuckle of bowel has been caught in its meshes. Sometimes sausage-shaped tumours may be palpated and percussed which are obviously caused by a loop of intestine bent sharply upon itself and glued down or surrounded by fibroid growth or caseating tubercular tissue.

Other tumours may be felt independent of any connection with the intestine ; these are nodular and more or less movable, and from their situation and feel can be diagnosed as enlarged or caseating mesenteric or retroperitoneal glands. When there is a moderate amount of fluid and the glands are large, as in the cases occurring in children, they may be demonstrated by 'dipping'—viz. by suddenly displacing the layer of fluid effusion by the finger-tips, which are thus brought into momentary contact with their smooth or nodulated surfaces. In some cases these glandular tumours constitute the only evidence of the abdominal tubercular condition, no peritoneal inflammation being present, in which case they may be taken as representing the typical form of mesenteric gland disease, under which heading they are referred to (p. 1041).

There may be signs of venous obstruction caused by the pressure of the inflammatory or tubercular tissue upon the portal system, as seen in the occasional enlargement of the superficial vessels of the abdomen and oedema of the lower extremities.

Allchin emphasises the importance of the diagnostic value of redness and inflammatory swelling about the umbilicus, which is sometimes perforated by the burrowing of pus along the foetal remains of the urachus and the establishment of a faecal fistula communicating with a perforated coil of bowel. This is a different condition from the non-inflammatory

retraction of the navel observed by the writer in the adhesive forms of chronic peritonitis, in which all the mesenteric attachments are simply shortened by contracting fibroid proliferation.

At any stage the symptoms and signs may be suddenly altered by the supervention of acute obstruction of the bowel or by the formation of an abscess in a caseous gland or in the tubercular tissue gluing together neighbouring coils of the bowel.

In the tubercular peritonitis which sometimes follows upon cirrhosis of the liver in adults the signs of ascites will be obviously modified by the succeeding peritoneal mischief.

The course of the disease varies considerably. In the *fibroid* form it is usually very chronic, and, unless obstruction of the bowel supervenes from adhesions, the malady may exist for years before wearing out the patient's strength by prolonged marasmus; in some cases complete resolution occurs.

In the *caseating* or *ulcerative* variety the course is seldom so protracted, but ultimately lardaceous disease supervenes, and as the bowel becomes involved in the amyloid degeneration an exhaustive diarrhœa may carry off the patient where no true tubercular ulceration of the mucous membrane has ever existed. The signs, symptoms, and course of the disease will be considerably altered by the tubercular process, which usually exists elsewhere in the body; thus life may be speedily terminated by the supervention of a tubercular meningitis.

Diagnosis.—The different points have been already emphasised in the description of the signs and symptoms. Owing to the extraordinary diversity of these in different cases, in arriving at a diagnosis the signs and symptoms as a whole must be duly weighed always along with the clinical history of the case; but too much importance should not be attached to a family-history of tuberculosis in a doubtful case. A strong point will be the discovery of lung or gland tubercle or any other focus of the disease elsewhere; occasionally the inoculation of ascitic fluid into the guinea-pig proves conclusive. Tuberculin injected into the patient may be sometimes resorted to with advantage in the diagnosis of a doubtful case.

Typhoid fever is eliminated with difficulty even in the chronic forms, and it is a not very uncommon event in every hospital-physician's experience to meet with cases of the typical tubercular disease after two or three months' illness which have been diagnosed and treated as typhoid fever from the first. It is often impossible to differentiate in the early stages, especially in the chronic examples of the disease which follow upon acute attacks of general tuberculosis, where the brunt of the attack falls upon the intestinal peritoneum. The absence of

the eruption and of Widal's reaction is of value in the exclusion of typhoid, even when the foul pea-soup motions are present ; but, upon the whole, the main reliance must be placed on a careful scrutiny or study of an accurately kept temperature-chart ; in the great majority of cases this is conclusive after two or three weeks.

Ascites may be the only sign present, and fortunately this, when the result of tubercular peritonitis, is more likely to occur in children, in which cases *Cirrhosis of the liver* may then be safely eliminated. In the adult, the history of alcoholism usually settles the diagnosis, but the frequency with which tubercular peritonitis supervenes upon hepatic cirrhosis must always be kept in mind.

Cancer of the peritoneum or of any of the viscera, especially when associated with liquid effusion, may produce a clinical picture closely simulating the tubercular affection when tumours are present ; but the characteristic omental tumour should not be mistaken for the lower margin of a nodular liver, as the resonance of the stomach and colon above it is usually capable of demonstration. The steadily progressive deepening of the wasting after a time, when taken in conjunction with the history and age of the patient, usually settles the diagnosis of cancer, though there may be a degree of pyrexia present suggestive of tuberculosis.

Ovarian cysts are sometimes closely simulated by a tubercular peritonitis, causing a localised or encysted accumulation of ascitic fluid. When such exists, it is, however, usually situated in the umbilical region, and careful palpation and percussion will usually reveal the presence of omental or mesenteric growths.

Where redness and swelling around the umbilicus occur, and especially when an intestinal fistula forms, the diagnosis is practically certain.

From *non-tubercular* chronic peritonitis the diagnosis is generally impossible till after careful watching of the case for a considerable period. The recognition of a non-tubercular cause, as appendicitis or pelvic inflammation, will help in the exclusion of the tubercular malady, as the discovery of tubercle elsewhere will usually clench the diagnosis in the opposite direction. In chronic non-tubercular peritonitis an inflammatory omental tumour is sometimes, though rarely, present ; but when present it is not associated with mesenteric gland-enlargement to any appreciable extent. It must always be borne in mind that extensive abdominal tuberculosis is not rarely found with little or no appreciable wasting, and with not only no fever, but with long periods of sub-normal temperature.

Prognosis.—Tubercle confined to the abdomen is more likely to end in recovery than tubercle deposited in any other

part of the body, the scrofulous adenitis of the cervical or axillary lymphatic glands excepted. Even in apparently acute cases complete resolution sometimes occurs. This was recently demonstrated in a number of cases of tubercular infection occurring amongst the isolated children of a large charity-school under the writer's observation. Fifteen girls were attacked with tubercular adenitis which had almost certainly been caused by the milk of tubercular cows, since four out of six of the animals supplying the milk were proved to be tubercular. One developed general acute peritonitis, with ascites and some mesenteric-gland enlargement, but complete recovery followed in this case after a period of three months, without operation. The outbreak was limited to those affected by immediately altering the milk-supply. In some the glands softened and suppurred, in others they were excised by a radical operation. A similar outbreak of tuberculosis was observed some years previously under the same circumstances, but, the cause not being recognised or traced for a considerable time till a fatal general acute tuberculosis had occurred, the glandular enlargements continued, and radical extirpation of them was followed by recurrences and death from phthisis in several cases.

The bacilli of tubercle appear to meet with some marked antagonism to their growth and development in the human peritoneal sac, and when the disease is detected early, and the environment of the patient promptly altered for the better, in a large percentage of cases recovery may be reasonably expected. In the fibroid form the prognosis is much better, than in the caseating or ulcerative variety, and in cases accompanied by much ascites the outlook after operation is decidedly better than it is in the dry cases. Obviously the prognosis is most unfavourable in all cases where phthisis and pleuritis are present. Even after apparent recovery the prognosis must be seriously modified when adhesions interfere with the functions of the stomach or bowels. A family-history of tuberculosis, though of little moment in the diagnosis of a doubtful case, becomes a serious item in the prognosis of the established disease.

Treatment.—The measures indicated in pulmonary and other forms of tuberculosis should be employed in all cases, as extra feeding, cod-liver oil, change of climate, open-air life, &c. The remarks already made about the part which uncooked milk plays in the causation of the disease should suggest to the physician that, as milk must enter largely into the diet of the tubercular patient, it should invariably be sterilised by the prolonged application of a heat below that of the boiling-point.

When the above measures fail after a few months' trial, the question of operation should be seriously considered and

acted upon, unless some serious contra-indication is present, as phthisis or kidney-disease. The operative measures consist in a simple abdominal incision, the separation of such adhesions as can safely be broken down, the evacuation of all ascitic fluid, and the removal of any purulent accumulations between contiguous coils of bowel.

The success of this simple procedure has been during late years demonstrated upon a large scale, and many authorities place the permanent recoveries as high as 75 per cent. Recovery has very often followed simple exploratory incisions made in doubtful cases of abdominal disease, which upon being found to be tubercular were stitched up and left alone without further manipulation. This result has also been frequently observed when there was no ascitic fluid to be let out. The *rationale* of the treatment is more or less a mystery, no definite explanation of its success being forthcoming. In many cases the extensive manipulation of the bowel in the separation of adhesions, the flushing-out of the cavity by antiseptic solutions, and the free exposure of the diseased membrane to the influence of light and atmosphere, have been considered each at times to be the main therapeutic agent in working the cure; but recoveries are frequent where none of these steps have been undertaken and where only the most trivial hyperæmia of the serous membrane could have been produced. In searching for an explanation of the success of this empiric treatment the writer has been led to the conclusion that, as the only constantly present step in the operative procedure, in addition to the simple incision, is the anæsthesia produced by chloroform, ether, &c., this may afford the clue. The bacillus of tubercle in the abdomen is certainly severely handicapped in its struggle for existence by the vital resistance of the tissues, and it would seem that a very little would turn the scale, in many instances, in the patient's favour. The permeation of the blood by the vapour of the anæsthetic may yet be proved to be the chief factor in effecting the resolution of the disease, and the subject has not met with the attention which its obvious importance demands.

Guinea-pigs inoculated with tubercle in the abdominal cavity die from the disease, even when frequently chloroformed, as witnessed by the writer; but these animals are so susceptible to the bacillus that such results cannot be taken as applying to the human patient.

The value of tuberculin-injections, exposure to the X-rays, and other methods of treatment suitable in lupus and phthisis, have been demonstrated in several cases. Giovanni advocates ecching of the hæmorrhoidal veins and counter-irritation of the abdominal wall by croton-oil; and this treatment has proved successful in some cases under the care of Fedeli.

PERITYPHLITIS. (See APPENDICITIS.)

PERTUSSIS. (See WHOOPING-COUGH.)

PETIT-MAL. (See EPILEPSY.)

PHARYNGITIS, ACUTE

Acute simple pharyngitis is known by a variety of terms, as Catarrhal Sore-throat, Simple Sore-throat or Angina Simplex, Hospital Sore-throat, &c., and, moreover, it is sometimes confused with the condition which starts in the tonsils, since these glands are usually also involved in the pharyngeal catarrh. The disease may be defined as an acute inflammation of the mucous membrane lining the pharynx, which usually involves the tonsils, uvula, fauces, and soft palate, and, less frequently, spreads to the nasopharynx and larynx.

Etiology.—The mild cases appear to be caused by exposure to cold; hence the disorder is called catarrhal sore-throat. The reduced temperature of the atmosphere probably has nothing to do with the mucous inflammation, unless by diminishing the resistance of the tissues it permits the development in the body of those micro-organisms present either in the atmosphere or normally in the human mouth. Nearly all forms of the disorder are infectious, and it is propagated by direct contagion, the microbe being disseminated in the fine spray produced during coughing and sneezing. Gout, rheumatism, and other poisons probably act by diminishing the natural tissue-resistance. Traumatic causes are rarely in operation; when they do act, it is the more sensitive membrane of the larynx which suffers most, as in the inhalation of steam and corrosive vapours.

The main etiological factor is therefore a microbic agent, which may under certain favourable circumstances cause the disease to appear in epidemic form. A specific organism probably does not exist, for the study of the etiology of the disease suggests that a number of very different bacilli and cocci may produce the condition, and it is also likely that the infection is usually or always a mixed one. The most severe cases are regarded and described as *septic*; in these streptococcus pyogenes is the most commonly met-with organism, but staphylococci of various kinds are also frequently discoverable in the secretion and mucous membrane. The

disease may be conveyed by the germs contained in foul sewer-gas and in badly ventilated hospital-wards; hence one very common variety is known as 'hospital sore-throat,' which is very infectious and may develop in every staff-official, nurse, or patient sleeping in a particular room or ward. Occasionally the disease appears to be produced in those in contact with scarlatina patients and who may develop no other sign of that disease. Watson Williams has recently drawn attention to the importance of recognising the *diplococcus pneumoniae* as the cause of pharyngitis; and French physicians recognise no less than five varieties of this affection—the suppurative, pseudo-membranous, follicular, erythematous, and herpetic.

Symptoms.—In mild cases there may be no constitutional symptoms, the only difficulty complained of being pain in the act of swallowing, and some dryness, tickling, and discomfort in the throat, causing a reflex cough or frequent attempts at the hawking up of a scanty adhesive secretion or the constant swallowing of the saliva.

In ordinary cases of smart degree there is a marked redness and some swelling of the pharyngeal membrane, which is dry and glistening at first; afterwards it becomes covered over with viscid secretion. The uvula is always red and swollen and the tonsils are involved in these cases. The constitutional symptoms, though not severe, are nevertheless characteristic of a general toxæmia; there is chilliness or distinct shivering, with headache, backache, and muscular or joint pains. The temperature rises in proportion to the malaise, the breath becomes foul, and the tongue coated. Constipation is the rule, appetite disappears, and often nausea is present.

Some hoarseness is soon evident, showing implication of the larynx. In a few days often the reflex cough gives way to a true bronchial irritation, with secretion from the tubes of large and middle size as the inflammatory process extends downwards. Before the appearance of the bronchial secretion a distressing sensation of tightness and rawness may be experienced along the trachea. Should the irritation extend in an upward direction there may be nasal catarrh or rhinitis, with implication of the Eustachian tubes, causing deafness, tinnitus, or earache.

In severer cases of a more marked septic type the openings in the tonsils are closed by an exudation of a cheesy consistence and of a yellow or green colour, the tonsillar glands themselves being deeply congested and swollen, the condition appearing undistinguishable from an acute follicular tonsillitis. It is to this latter variety of pharyngitis that the term *ulcerated sore-throat* is often wrongly applied, since usually no abrasions of the surface are present. The lymphatic glands are some-

what enlarged and tender, the jaws are stiff, and swallowing is difficult or painful.

In the still more severe examples of septic pharyngitis, which are described as of the *phlegmonous* type, the membrane is deeply congested and livid, the tonsils acutely inflamed, and there is great œdema of the entire pharynx, which tends to rapidly spread to the larynx, causing aphonia, dyspnœa, and threatening asphyxia, with total inability to swallow. This condition is caused always by streptococcus infection, and has been described as *erysipelas* of the throat. It is sometimes associated with erysipelas of the face, and occurs during scarlet fever and diphtheria in broken-down debilitated patients and chronic drunkards. The inflammation usually ends in suppuration and sometimes in gangrene, and the lymphatic glands are deeply involved.

When this phlegmonous type starts in the floor of the mouth or in the deep cervical fascia under the lower jaw it extends to the pharynx and larynx, and is recognised as *Ludwig's angina* or *submaxillary cellulitis*.

Unless prompt surgical measures be employed, death from suffocation caused by the extensive œdema is very liable to occur. When the patient survives the danger of asphyxia the deep cellulitis may extend downwards beneath the sternum, and an acute purulent pericarditis or pleuritis may follow, or a general pyæmic state may be induced. The constitutional symptoms in these grave types of septic pharyngitis are marked from the initial rigor by extreme prostration and embarrassment of the heart and respiration. The urine usually is albuminous and often contains some sugar.

Scarcely less dangerous is the variety known as *Senator's phlegmon*, in which the streptococcic inflammation remains confined to the tissues of the pharynx and soon renders swallowing impossible; suppuration rapidly supervenes and death from asphyxia is very liable to occur.

Retropharyngeal Abscess is the name given to a collection of pus which gathers in the connective tissue between the back of the pharynx and the front of the spine. It is mentioned in this connection, as it is not uncommonly the result of a septic pharyngitis originating in ill-nourished infants during an attack of one of the acute exanthemata. In most cases it arises from an infection of the retropharyngeal glands of Henle; in older subjects it is more likely to be the result of caries of the cervical vertebræ. The collection of pus is easily recognised by its projection into the pharynx; in the early stages the symptoms are those of a smart pharyngeal inflammation, causing dysphagia and also hoarseness from pressure on the larynx, with some fever, and, at a later stage, aphonia, and sometimes stridor.

Diagnosis.—There is not likely to be any difficulty in the diagnosis of an acute inflammation of a mucous membrane which is visible and accessible to the touch. *Scarlatina* may first show its appearance by the vivid redness of the pharynx, but the severity of the constitutional symptoms and high range of temperature only simulate the graver septic forms of pharyngitis, which, however, are devoid of the characteristic scarlet eruption. The phlegmonous types are recognised by the presence of the streptococcus pyogenes which causes erysipelas; and *diphtheria* is eliminated by the absence of its characteristic false membrane, which never appears on the tonsils in the form of the yellowish specks projecting from the lacunæ and separated from each other by swollen tonsillar tissue. In doubtful cases the detection of the Klebs-Loeffler bacillus will always settle the diagnosis of diphtheria.

Prognosis.—Mild cases of simple pharyngitis run their course in less than a week, always ending in complete resolution. The severer septic variety known as hospital sore-throat may be of slightly longer duration, but its prognosis is always good. The outlook in the phlegmonous cases is grave, the mortality being very high. The gravity may be gauged by the cardiac condition and the amount of laryngeal œdema.

Treatment.—The simple cases require little medication; a smart saline purge, followed by a diuretic, of which salicylate of soda is the best, usually relieves the constitutional disturbance. The best routine local treatment is the spray of a 1 per cent. solution of pure carbolic acid, to which a little cocaine may be added when pain is prominent. In the mild septic cases this local treatment is usually sufficient; but for internal administration nothing gives so much relief as small and frequently repeated doses of antipyrin.

In the phlegmonous types tincture of iron in full doses has long been the favourite drug with surgeons; it should be combined with quinine, pushed till mild cinchonism is experienced. Antistreptococcic serum should be hypodermically administered in all cases. Tincture of aconite, which is often employed in the mild cases to reduce temperature, should never be employed in the grave cases owing to the liability to cardiac failure. Symptoms of laryngeal embarrassment as they arise must be met by prompt surgical methods, as scarification of the œdematous tissue about the entrance to the larynx, tracheotomy, or laryngotomy.

Feeding is of vital importance from the beginning; the most concentrated and nourishing semi-liquid soups and broths should be persistently administered by the mouth when possible, or by the rectum when swallowing becomes arrested. Strychnine hypodermically should be given as

soon as cardiac weakness shows itself, and oxygen-inhalations are necessary as soon as any degree of lividity appears. All local abscesses should be evacuated by a free incision as soon as fluctuation can be detected; often in deep cellulitis it is imperative to make free incisions to relieve tension without waiting for the usual signs of suppuration.

PHARYNGITIS, CHRONIC

This is a chronic inflammation of the mucous membrane lining the pharynx, sometimes throughout its entire extent from the beginning of the œsophagus below to the nasal cavities above. Various clinical types of the disease have been described, the two most commonly recognised forms being the *Granular* or *Hypertrophic* and the *Dry* or *Atrophic*; but such division must not be hard and fast, as both varieties of chronic pharyngitis may be seen in different parts of the same throat at the same time. Some authorities recognise but one form under the title *Chronic Catarrhal Pharyngitis*; but, as pointed out by Horne, these conditions are all merely phases of the same diseased process.

Etiology.—The chronic form is supposed sometimes to be the sequel of one or more attacks of the acute type of pharyngitis. Much more frequently it is an essentially chronic inflammatory process from the first, starting in a chronic nasal catarrh (atrophic or hypertrophic rhinitis).

Alcoholism is a common cause, in which case the main factor in the pharyngeal condition is probably a chronic gastritis, which produces the characteristic morning nausea giving rise to incessant hawking and strain upon the pharyngeal muscles and tissues. Chronic dyspepsia appears to act in a similar manner.

Tobacco, and especially cigarette-smoking, is recognised by most authorities as a fruitful source of the disease. The writer has been struck by the frequency with which the typical condition is met in non-smokers and the comparative rarity of it in those who consume large amounts of tobacco, and he is satisfied that nicotine plays, if any, but a small part in the etiology of the disease.

Excessive use of the voice is too often regarded as a main factor, since chronic pharyngitis is so often seen in clergymen, public speakers, street-criers, and singers. The use of the voice in loud speaking in ill-ventilated rooms overheated by gas, and especially when the atmosphere is laden with dust or irritating vapours, certainly tends to induce changes in the pharyngeal membrane, but probably never unless in association with laryngitis.

The comparative rarity of the condition in women would seem to prove that the abuse of tea plays no part in its etiology. The most obstinate forms are often associated with the gouty or rheumatic diathesis, and are not infrequently met with in those suffering from chronic eczema and valvular disease with venous congestion. It is rare in those living in rural districts, whilst common in cities, especially amongst hard-working sedentary clerks and individuals who constantly over-tax their mental and physical powers. It is seldom seen in labourers, and is rarely met with till after puberty or near to middle-life.

Symptoms.—The most constant evidence of the disease is a sensation of irritation about the fauces, causing continual efforts at hawking up secretion from the throat; often a short tickling cough is the predominant symptom. The cough is almost distinctive and suggests the idea that it is a *habit*-cough which could be easily controlled or inhibited; it is usually unaccompanied by secretion.

Some impairment of the voice or, more commonly, a painful sense of fatigue after speaking, is complained of, though the larynx shows no signs of change. It is this sensation of pain after speaking and the presence of vocal impairment which has given the name of '*clergyman's sore-throat*' to the affection, though this term is sometimes also applied to a chronic laryngitis. When one considers the frequency with which typical chronic pharyngitis is discovered in a routine examination of patients who complain of no throat-symptoms, the conclusion seems often justifiable that the prolonged use of the voice merely demonstrates the presence of the throat-affection and has no part in its production or causation.

On inspection, the pharyngeal membrane is found red and irritable, the slightest touch inducing nausea or retching. It may be smooth, glistening, and dry, as in the atrophic variety, with a few patches of thin mucous secretion adherent at places, constituting the condition recognised as *pharyngitis sicca*. More frequently it is distinctly thickened, and the uvula and tonsils swollen and covered with moist mucous secretion; the nasopharynx is involved in the swelling and the Eustachian tubes more or less implicated, as seen in some interference with hearing. The most characteristic signs are observable on the posterior wall, where numerous small grey or red flattened granulations are visible. These are caused by the hyperplasia of the diffuse lymphadenoid tissue, and are not necessarily of follicular origin, as implied in the old name of the condition—*follicular pharyngitis*—a name applicable only to those cases where the follicles caseate and form small ulcers.

The term *granular pharyngitis* is preferable, even when a follicular tonsillitis is present. The granulations vary in

size, the majority being not larger than a line in diameter ; small dilated veins are usually associated with them ; sometimes minute ulcers are visible, and the slit-like openings into crypts of the tonsils are often blocked by thickened pellets of caseating exudation.

The disease runs an essentially chronic course, especially in neurotic patients, who give way to incessant hawking and coughing in order to clear the throat—a habit which certainly tends to perpetuate the condition indefinitely. A common and almost constant clinical feature in the disease is the liability to acute or sub-acute attacks from some passing phase of gastric or other disturbance, or bronchial catarrh which induces frequent coughing.

Diagnosis.—*Syphilitic* ulceration is excluded by the small size of the ulcers when such are present. The syphilitic ulcers are oval, with sloughing bases and sinuous outlines ; syphilitic mucous patches are also always larger than the granulations of chronic pharyngitis, and, moreover, other signs of the specific affection are always to be found elsewhere.

Tubercular ulcers are more extensive than the follicular ulcerations seen in granular pharyngitis ; they have eroded worm-eaten edges, are often circular, always pallid, very painful, and generally start from the base of the uvula.

There is a *chronic hyperplasia* of the pharynx, described by Semon and Brown Kelly, which causes enormous thickening of the mucous membrane, involving the uvula, soft palate, faucial pillars, sometimes invading the larynx and blocking up almost completely the opening into the nares. The amount of fibroid proliferation and thickening far surpasses the swelling observed in chronic pharyngitis.

Pharyngomycosis is a rare condition hardly liable to be mistaken for granular pharyngitis ; in it small white excrescences appear mostly about the base of the tongue and pillars of the tonsils. These resemble leucoplakia somewhat, and are studded over apparently perfectly healthy mucous membrane. The microscope reveals hypertrophy or cornification of the epithelial layer, and hence the disease has been called *Keratosis pharyngis* ; but as leptothrix filaments are often present it has been also regarded as a mycosis. It is an innocent, very chronic, condition of little clinical importance.

Treatment.—As the disease is so frequently found in debilitated individuals, the first indication is to correct any departure from the general standard of health and to give the pharynx as much rest as possible. For this latter desideratum straining of the voice and hawking or coughing should be guarded against. Local applications of strong nitrate of silver solution or zinc chloride are very valuable as astringents and caustics, whilst a weak carbolic spray (1 in 100) reduces

the hyper-sensibility or tickling and diminishes the attempts at throat-clearing.

The most efficacious treatment of the granulations and hypertrophied masses of lymphadenoid tissue consists in the free use of the electric cauter. Diseased tonsils, when hypertrophied, should be removed by the guillotine, or their bulk reduced by puncturing at numerous points with the cauter; often the elongated uvula can be removed with much advantage. In the dry atrophic form great benefit may be obtained by the continual use of the nasal douche, or preferably by the patient sniffing up a weak solution of borax from the palm of the hand, so as to thoroughly flush out the nasopharynx many times during the day.

PHLEBITIS

Inflammation of veins is chiefly of surgical importance. Under 'Thrombosis' are mentioned the serious forms of portal phlebitis which come under the province of the physician. Inflammation of the lining membrane of the veins is liable to occur in small-pox, typhoid fever, anæmia, and various septic and cachectic conditions, and the complication is referred to in the description of each malady. Paget described a form of recurrent phlebitis of the veins of the extremities which he attributed to gout. Briggs, in his recent published cases, failed to find evidence of the uric-acid diathesis, and regards the pathology of these cases as depending upon sclerotic changes in the venous wall akin to the fibrosis seen in the small arteries. The main result of the inflammatory action is the formation of a thrombus, which, being detached, may cause serious embolic plugging in distant parts or upon reaching the heart may cause sudden death.^{F 7}

PHOSPHATURIA

The presence of phosphates in the urine in excess is dependent upon several very different conditions, and often has little clinical significance. Phosphoric acid to the amount of from 15 to 80 grs. is excreted daily in the urine, considerably less than the half of which is in combination as *earthy* phosphates. These occur as acid calcium phosphate, tri-calcic phosphate, di-acid calcic phosphate, ammonio-magnesium phosphate, and, less frequently, as neutral magnesium phosphate. The *alkaline* phosphates in the urine are very soluble salts, and do not, like the earthy phosphates, appear as deposits; they consist of acid potassium and sodium phosphates.

The source of phosphoric acid is mainly the food ingested ; a small quantity comes from tissue-metabolism, phosphorus being an important constituent of nucleo-proteids and lecithin. A diet rich in animal food produces a larger excretion of phosphates than a purely vegetarian régime.

There is constant confusion of thought as regards what constitutes the condition termed phosphaturia or phosphuria. Phosphatic urine is erroneously regarded as evidence of phosphaturia ; it may exist when the daily output of phosphates is diminished. Urine which is alkaline from any cause deposits its earthy phosphates ; hence the mere presence of these may have no clinical significance, save that pertaining to the alkalinity. Nor is the form of the deposit of importance. If the urine be markedly alkaline, the amorphous phosphate of calcium is thrown down ; if it be almost neutral, the crystalline phosphate is deposited ; whilst if the urine be highly acid, it may be laden with lime-salts, which continue in solution, the oxalate only being sometimes deposited. Urine which is normal when excreted by the kidney may undergo ammoniacal fermentation in the bladder, in which case the earthy phosphates, usually in the form of the triple or ammonio-magnesium phosphate, are deposited.

It is thus evident that the mere presence in the urine of the earthy phosphates, even in apparently large amounts, should not be mistaken for phosphaturia ; this, in order to constitute the so-called 'diathesis,' must be defined as an elimination of phosphoric acid in combination, as phosphate of lime, in such abnormally large amount as cannot be explained by dietetic fluctuations. The normal standard for the twenty-four hours' elimination, as already stated, varies greatly (from 1 to 5 grms.), the average being taken as half this latter figure, or about 36 grs., the greater portion of which in the normal condition is in combination with sodium.

The clinical signification of the increased daily output is regarded by some authorities as of considerable importance. Tessier has given the name *phosphatic diabetes* to the condition wherein there is marked increase of phosphates eliminated and where the proportion of alkaline to earthy phosphates is reversed. In the normal state the alkaline phosphates much exceed the earthy ones in amount, but in the condition just mentioned the lime phosphates may quadruple the potassium and sodium phosphates. The urea is usually increased in these cases ; the urine is commonly acid, and may therefore show no deposit or cloudiness ; it is pale and very copious, and throws down a large deposit of phosphate of lime upon the addition of ammonia or liquor potassæ.

This is the state described by Dickinson as occurring in nervous, excitable, middle-aged males, associated with mental irritability, sleeplessness, depression, arthritic pains, and

sometimes fugitive numbness of the legs and subjective head-sensations, as dizziness and tinnitus. The tongue is flabby coated, and anæmic, and is made worse by the administration of alkalies and improved markedly by acids. Emaciation is common. Ralfe has observed that many of these patients become phthisical, and some develop saccharine diabetes. Occasionally only is the urine alkaline and cloudy, but often heating the clear urine causes a copious deposit of calcium phosphate, which instantly disappears upon the addition of a mineral acid.

There is much difficulty in the interpretation of the pathology of the phosphatic diathesis, many authorities considering that it is indicative of the breaking-up of the nucleo-proteids and lecithin in abnormal metabolism. As held by Edlefsen, it may be regarded from the same point of view as the increased amount of nitrogen or urea in the abnormal metabolism which occurs in the other tissues during various conditions, as the febrile state, &c. This view is supported by the authority of Dickinson, who shows that phosphaturia occurs during some strain or stress of hard intellectual work in individuals of 'nervous, mobile, and hypochondriacal temperament,' though he, like others, failed to see what lime has to do with the brain and how it can be an appreciable product of its waste; nevertheless, he regards the excretion of an abnormal amount of earthy phosphates as a common clinical manifestation of many forms of nervous irritation, and the demonstration of such in the urine by precipitation with liquor potassæ he takes as a valuable guide to the therapeutics of the diseased condition of the patient.

Excess of earthy phosphates also occurs in saccharine diabetes and in tubercular disease, especially when the meninges of the brain are involved. Diminution in this excretion is commonly observed in all structural renal diseases.

Diagnosis.—The main feature of clinical importance is the increased elimination of the *earthy* phosphates when the total daily output of urine is examined; increase of the alkaline phosphates may be taken as of little or no significance. All the forms of neurotic dyspepsia, by diminishing the acidity of the urine, may cause deposition of lime-salts, which must not be accepted as indicative of the phosphatic diathesis. In the same way, every other cause which diminishes the acidity of the urine must be recognised, especially the structural diseases retarding the discharge of the renal secretion from the bladder. In rickets and osteomalacia the excess of lime-salts in the blood is excreted by the bowel, and hence, as proved by Barlow and Jacobi, excess of earthy phosphates is not observed in the urine.

Prognosis.—This is usually favourable, but when the abnormal amount of phosphoric acid greatly exceeds that of

the urea, as in the cases investigated by Ralfe, and falling under the severe degree of phosphaturia to which the term 'phosphatic diabetes' is applicable, the prognosis is serious; most of such cases terminate fatally in phthisis, diabetes, or other debilitating condition.

Treatment.—The nerve or mental strain should be relieved, and every departure from the healthy standard remedied, as far as possible by judicious rearrangement of the dietary and of the hours of labour and rest; open-air occupations and recreations should be recommended. The drugs which give best results are the mineral acids, especially the nitro-hydrochloric in conjunction with strychnine. Opium, morphine, and codeine may be employed, as in diabetes, when the amount of urine is excessive.

PHTHISIS

For all practical purposes the term 'phthisis' may be defined as *Pulmonary Tuberculosis*. Its popular name—'consumption'—has the same origin as the word 'phthisis'; both imply wasting or emaciation, which is the chief obvious result of the disease.

The term 'phthisis pulmonalis' is employed sometimes to prevent confusion with such conditions as abdominal or intestinal tuberculosis, often called consumption of the bowels.

As implied in its name of pulmonary tuberculosis, all authorities now recognise the unity of the different varieties of phthisis—all are the result of an invasion of the pulmonary substance by the *bacillus tuberculosis* of Koch.

The disease is the most important of the many morbid processes which affect the human race, since at least one-seventh of all the deaths occurring throughout the world is due to tuberculosis. Of the total cases of fatal tuberculosis, in about thirteen out of every fourteen the pulmonary organs are affected, and the affection is in the great majority of instances primary.

Etiology.—The causation of phthisis is, in a broad sense, that of the other forms of tuberculosis and has already been mentioned in discussing the etiology of abdominal, meningeal, laryngeal, renal, and other varieties of the disease, and will be again referred to under 'Tuberculosis.' Nevertheless, the enormous importance of the subject of pulmonary tuberculosis warrants more than a passing reference to the etiological factors which are at work in the introduction and development of the bacillus of tubercle in the pulmonary organs.

The disease is distributed over the entire habitable globe, being, however, most prevalent in the densely inhabited regions. Climate, upon the whole, must be said to play a minor rôle in its causation, since it flourishes in the cold,

temperate, and hot regions of the earth. It is comparatively rare in high altitudes, and apparently comparatively more frequent in cold moist climates. The disease is prevalent amongst all peoples, though the Jews are held to be less vulnerable than other races; this is supposed to be explicable by their obedience to their superior sanitary code, especially as regards the supervision of their meat-supply. The Indians of North America—especially those, according to the writer's personal observation, inhabiting the Reserves in the Dominion of Canada—suffer heavily from phthisis and glandular tuberculosis. The negro is also highly susceptible, as are apparently all the dark-skinned races, especially when these are brought under the influences of European civilisation. Of far more importance, from the etiological standpoint, than any of these considerations is the deleterious influences which are brought into operation by the congregation of large numbers of individuals in cities, especially where unhealthy occupations are pursued in dust-laden and ill-ventilated factories and workshops. These influences, as will be presently seen, are supposed to be chiefly potent by reason of the facilities which they afford for the spread of the disease from infected individuals to those brought into contact with them.

Sex has little influence when the tuberculous of all ages are included in statistics, though females suffer much more than males between the ages of five and thirty-five years; and male children during the first year of life are twice as frequently affected as female. After thirty-five the death-rate amongst males steadily rises and always exceeds that of the female mortality.

Age as an etiological factor shows its influence by the highest mortality being recorded between the ages of twenty and thirty-five; but all periods of life, from early infancy to extreme old age, supply their quota of cases, though the disease is comparatively rare between the first and fifth year and after seventy. Many authorities maintain that the varying incidence of the disease at different periods is entirely a matter of exposure to infection, and that age as an etiological factor in the strict sense should be ignored.

Hereditry plays no part in phthisis—that is, in the strict sense of the tubercle bacillus being transmitted in the seminal fluid or by the placental blood; but hereditary predisposition cannot be doubted, though Cornet contends that there is no evidence forthcoming to prove any transmission of hereditary disposition. Most physicians can recall instances of large families which had scattered early in life over wide distances and after many years the different members in distant regions succumbed to phthisis. Cornet regards the reference to such examples as 'rhetorical hyperboles,' explaining the fact to his own satisfaction by the theory of the children being

already tuberculous before their separation, or that further opportunities for infection were not excluded. The writer can recall instances in which one young member after another of a large family in a small Irish homestead was compelled to push out abroad into different climates, and where each died of phthisis at middle-age or beyond it. In one remarkable case twelve members succumbed in this manner, though several of them had left home before their successors were born, and some lived to the age of forty. In some examples of this kind, as in the last-mentioned case, the disease may resemble a family-disease and not an hereditary one, in so far that both parents may survive their entire offspring and die at extreme old age, the predisposition being transmitted, as in the case of bleeders, by one or other or both of them. Though the fallacies are numerous and obvious when statistics are quoted to prove the existence of hereditary predisposition in such a common disease as phthisis, nevertheless the fact seems to be demonstrated by a perusal of the family-history of many patients. Those who hold the opposite view attach too much importance to the sole consideration of infection, and fail to recognise the practical ubiquity of the bacillus because only a small proportion of the individuals living are infected by the organism. If the enormous amount of tuberculosis which always exists in the cattle affording the milk and meat supply of the world is realised, it becomes obvious that the bacillus must be regarded as practically ubiquitous, and some process of immunity must be in operation, else the human race would soon disappear. Hence this lack of immunity which physicians style hereditary predisposition causes the minority to succumb, it being always taken for granted that probably no individual is immune to a colossal dose of the microbes. The question whether such immunity is inherited or acquired cannot be settled at the present stage of our knowledge, nor need time be spent in the discussion of the theories which have been invented to explain it. It may be well, however, to accept as a working hypothesis that the congenital predisposition to phthisis may be the result of, or a manifestation of, some deficiency in the mechanism of phagocytosis, and that the acquired immunity may *within certain limits* be explicable on the hypothesis which is usually accepted as an explanation of serum-therapy. Professor Lindsay states that a definite family-history of phthisis is obtainable in about one-third of all cases coming under observation, and, whilst acknowledging that the significance of a family-history has been exaggerated in the past, he rightly considers that it is a great error to regard it as of no significance.

Falling under the same category as predisposition or lack of immunity is the important group of etiological factors

which includes pregnancy, hyper-lactation, under-feeding, over-work, the exhaustion caused by various diseases as measles, whooping-cough, influenza, typhoid fever, scarlatina, diabetes, hepatic cirrhosis, and syphilis. That any or all of the above conditions may eventuate in phthisis cannot be disputed, and the above-mentioned hypothesis may be used to explain their influence. These factors diminish the natural resistance of the body and permit of the development of the bacilli, which are perhaps always finding their way into the human organism in small numbers. Trauma, which is an obvious cause of tuberculosis of the testicle and spinal column, acts in the same manner; and this appears to be a highly probable or almost certainly true explanation of the phthisis which follows simple pleurisy or, more rarely, pneumonia. Severe contusion of the chest-wall or a previous pleural inflammation lowers the local resistance to the development of the organism within the chest. In this association it is interesting to remember that in predisposed individuals, in whom we may assume that there is some congenital diminution of the normal resistance, phagocytic or humoral, there is commonly evidence also of ill-formed or imperfectly developed thorax.

When all the above etiological factors have been considered, there remains the chief and, according to some authorities, the only real causative agent worth considering—viz. the process of infection by which the specific bacillus gains its entrance into the lung. The most important point in this connection is that pulmonary phthisis in the vast majority of instances is *primary*—a fact which strongly suggests, but does not prove, that the bacillus gains its entrance into the lung by the air-passages.

Experimental results until recently have appeared conclusive upon this point. The inhalation of pure cultures of the bacillus or of the dried and pulverised sputum of phthisical patients, when administered to animals under conditions as nearly as possible imitating those which maintain in the infection of human beings, always gives the same result. Tubercles are formed in the lung; these caseate, soften, and produce cavities, the bacillus being always present. The dried sputum of phthisical patients in the dust-laden air of populous cities is always a factor to be reckoned with, though probably very few of the microbes reach the terminal bronchioles and alveoli, the majority being arrested in the upper air-passages. It is proved beyond doubt that the bacillus of tubercle can pass through the normal mucous membrane of the respiratory and intestinal tracts; and since *post-mortem* examination has demonstrated the presence of coal-dust in the alveoli of individuals breathing air charged with it, there appeared to be no difficulty until recently in

accepting the possibility of phthisis being communicated in this manner.

The influence of sunlight in destroying the virulence of the bacilli, their comparatively small numbers in the air, and the activity of the natural protective agencies inside the body, account readily for the fact that, after all, only a fraction of the inhabitants of a large city suffer from the established disease. In considering the probabilities of a successful infection by the bacillus, the main question so often lost sight of is that of the *dosage*. This is proven by the fact that the disease is much more liable to be acquired by those who breathe the air of ill-ventilated dwellings in which phthisical patients have resided. In the writer's opinion, the danger of infection from the inhalation of air containing dried pulverised tubercular sputum is infinitely less than that of the inhalation of the fine spray which phthisical patients eject from the open mouth in the act of coughing. This germ-laden spray is very different from the exhaled air in quiet breathing, which is probably entirely devoid of bacilli, and, moreover, it may be ejected to a distance of several feet in severe coughing. When it is remembered that a patient suffering from advanced pulmonary phthisis may daily expectorate a quantity of bacilli equal in number to the entire human population of the globe on which we live, it is obvious how serious the problem of possible infection becomes.

Notwithstanding the results of experimental research and the fact that phthisis is so constantly a primary tuberculosis of the lungs, many authorities maintain that the disease is caused by bacilli introduced into the body in the food and carried to the lung by the blood or lymph stream. Von Behring believes that phthisis in the adult results from the tardy evolution of an intestinal tuberculosis contracted during early life by feeding on tubercular milk. His views are supported by the recent experiments of Calmette and Guérin, who accept his conclusions, and consider that the penetration of air-borne bacilli into the lungs is difficult, if not impossible. Ingested bacilli pass readily through the intestinal epithelium without causing breach of surface, and, passing into the chyliferous vessels, they are seized upon by the polynuclear leucocytes, which they deprive of amoeboid motion. These paralysed and infected cells find their way into the venous circulation and become arrested in the pulmonary capillaries, which they block, the bacilli remaining as foci in the lungs; but if the cells escape through the pulmonary sieve, meningeal or other forms of tuberculosis result.

Calmette and Guérin, moreover, go further than Behring, and state that, as the results of their experiments of suckling kids by goats whose udders had been inoculated with

human, bovine, and avian tubercle, the bacilli were retained for varying periods in the mesenteric glands of the kids, and ultimately caused pulmonary lesions, whilst in older animals the bacilli almost immediately infected the lungs. From this research they are led to affirm that 'pulmonary tuberculosis at any age may have a *recent* intestinal origin.'

The inhalation of dried sputum causing pulmonary tuberculosis in animals must therefore be accepted with a certain amount of caution, since it is always probable that the animal swallows some bacilli in his saliva, which, reaching the intestine, may take the route indicated above and eventuate in phthisis. This, in Calmette's opinion, is proved by the fact that anthracosis of the lungs is not produced in animals by making them breathe an atmosphere of soot, so long as precautions are taken to prevent their swallowing the accumulations in the nasopharynx.

Should these interesting results be corroborated, the portal of the intestinal epithelium must be regarded as the main etiological feature in the production of phthisis; in which case the interest in the most serious problem affecting the well-being of mankind shifts from the consideration of infected air to the infected milk-supply. It may be accepted that tubercular infection is possible even when the udder of the diseased animal remains apparently free from tubercle. Notwithstanding the strong belief and statements of Koch, the majority of pathologists are satisfied that bovine tuberculosis is communicable to man, and it has been demonstrated beyond doubt that human tuberculosis is easily transmitted to animals.

Under 'Peritonitis, Tubercular,' p. 1285, the writer has stated the reasons, derived from clinical observation, for his belief in the important *rôle* played by the bovine bacillus in milk in causing infection of the mesenteric and cervical glands, which sometimes ends in pulmonary tuberculosis.

It must be also mentioned that Ribbert, previous to the researches of Calmette and Guérin, maintained that the bacilli reached the bronchial lymph-glands; having in the first instance, he believed, been admitted by inhalation they passed through the pulmonary tissue into the lymph channels and glands, from which latter situation they were carried *by the blood-stream* to the apices of the lung—a view which to some extent harmonises with the hæmatogenous route selected by the bacilli in their progress from the mesenteric lymph-glands.

Calmette and Guérin in a second series of experiments have verified their former results, and conclude that 'the so-called primary pulmonary tuberculosis of the adult is most frequently of intestinal origin; and that, of all the modes of infection, infection by the digestive tube is the most effec-

tive and corresponds the best with the normal conditions of natural infection.'

The tonsils and the shallow cavities of necrosed teeth may become the culture-grounds for the bacilli, or the latter may find a direct entrance into the system through these portals. It is very striking how frequently phthisis attacks individuals whose teeth are necrosed and where granulation-tissue exists in their cavities.

Morbid Anatomy and Pathology.—The bacillus of tubercle on its introduction to the tissue of the lung causes histological changes akin to those produced in other organs or tissues. The outcome of these changes is the formation of the so-called 'tubercle,' which is produced by the specific irritation of the bacilli or of their chemical toxins. The first stage in the process is the production of epithelioid cells, which by rapid division multiply; these, together with lymphoid cells distributed round the periphery, and often one or more giant-cells (with numerous oval nuclei) in the centre, constitute the tubercle or tubercular nodule typical of miliary tuberculosis. The bacilli lie between the cells, often they are seen in their interior; in the giant-cells they may be found in large numbers. The reticulum or stroma is comparatively insignificant and is derived from the remnants of the original fibrillar stroma of the tissue. In bulk the tubercular nodule is usually about the size of a millet-seed, often not exceeding 1 mm.; it is firm and grey in colour. After reaching its full active development, caseation sets in, commencing in the non-vascular centre; the colour changes to a yellowish-white, the cells break up, their protoplasm vanishing, nothing being left but a hyaline mass with fatty granules and bacilli. Later on the caseated mass softens and becomes converted into pus. In the cheesy stage of the tubercle it may be dead in the centre, whilst from the periphery the bacilli are actively increasing and invading the neighbouring pulmonary tissue with newly forming nodules, which in their turn caseate and soften, so that large cavities are formed in the lung-substance.

If the pulmonary infection has been caused by an invading host of bacilli *via* the blood-stream from some caseating focus at a distance, both lungs, with their pleuræ, may be involved in an acute general miliary tuberculosis. In typical chronic phthisis the bacilli in small numbers, whether admitted by the respiratory or intestinal tract in the first instance, reach the terminations of the bronchi in the alveoli, and there set up their specific irritation, which eventuates in caseation. In some autopsies the cheesy nodules may be found encapsuled and their contents often infiltrated with lime-salts; in others a firm cicatrix only marks the site of the infection, complete resolution having taken place. In patients dying of chronic phthisis the changes in the lung vary considerably; two

types are recognised, though in all cases the changes are to be regarded as mere variations of the above-described forms of invasion, irritation, proliferation, caseation, and softening. When the bacilli reach the lung by the lymphatics the resulting tubercles are laid down in the direction of the peribronchial lymph-channels, constituting the changes described as characteristic of tubercular *peribronchitis*, in which the interstitial tissue of the lung is chiefly involved. When the bacilli are supposed to reach the alveoli and bronchioles from without in the inspired air the mucous surface is first involved, and the changes recognised as *cheesy pneumonia* or *tubercular bronchopneumonia* are met with, in which an extensive fibrinous exudation, with proliferation of leucocytes and shedding of the alveolar epithelium, renders certain lobules or portions of a lobe of the lung solid and airless. In all cases the final results are practically the same; as caseation is followed by softening excavation occurs, neighbouring nodules coalesce, their softened contents burst into a bronchus and are expectorated. The bacilli being freely poured into the tubes become foci for further infection, and by the lymph-channels and blood-vessels they may be carried to distant parts of the lung, or even by aspiration they may reach the bronchi of the sound organ.

In the early stages of phthisis the disease is the result of a pure infection caused solely by the bacillus tuberculosis, but the opening up of the small cavities or vomicæ into the bronchi introduces new elements into the destructive process by the admission of various pathogenic organisms from without. As long as a cavity is encapsuled or shut off from the bronchial surface pus-formation may proceed in it through the inflammation caused by the highly irritating toxic products manufactured by the bacilli. The most common and, as held by most authorities, the invariably present organisms are streptococci in cavities communicating with bronchi; streptococcus pyogenes is always abundant, though many other cocci, diplococci, and bacteria are also found. These organisms reinforce the tubercle bacillus in its destructive action upon the pulmonary tissue; small cavities coalesce from the destruction of their partitioning walls, and a considerable portion of the lung becomes honeycombed, with numerous excavations in direct communication with each other. More rarely one large cavity occupies the entire upper lobe of the lung. In the smaller vomicæ of the less chronic type of case the process of excavation may be generally demonstrated as occurring from the beginning in the breaking-down of the wall of a bronchus; such cavities have no definite lining membrane: their walls are ragged, being constituted by necrosing or ulcerating tissue which had previously undergone caseation.

The typical cavity of *chronic* phthisis is lined by a pus-secreting membrane which is often highly vascular. Through the cavern, bands of tissue are found extending from one side to the other; these trabeculæ on dissection are often found to contain large blood-vessels and sometimes bronchi. Where hæmorrhage has preceded death the ruptured vessel may be found in these trabeculæ, and often aneurismal dilatations are evident upon the smaller vessels supplying the vascular limiting membrane or projecting into the cavity from the larger arteries which traverse it. These aneurisms, which are usually minute, are a common source of hæmorrhages; when they are large and in direct communication with a moderately sized vessel a rapidly fatal hæmoptysis may be caused by their rupture. The necrotic process, however, by the time it comes to involve the arterial walls, is, fortunately, usually rendered bloodless by the previous blocking and obliteration of the lumen of the vessel. Even in the more acute cases, when the walls are sloughing or gangrenous, the blood-vessels may be found more or less completely occluded and often the seat of tubercular deposit.

The contents of the cavity correspond in the main to the matter which is expectorated; pus is abundant, yellow caseating masses and white particles are present, the latter consisting of almost pure cultures of the specific bacillus. Lung-detritus, in which yellow elastic tissue may be discovered, and abundant pathogenic organisms are also nearly always present.

The diseased process usually starts in the apices, and is always found in a more advanced stage in this region when a considerable proportion of the entire upper lobe has become affected. No really satisfactory explanation of the predilection for this site by the bacillus is yet forthcoming. Those who hold to the origin of phthisis being always due to the inhalation of bacilli regard this peculiarity of its localisation in the lung as proof that the disease has gained entrance with the air-stream, inasmuch as the germs are permitted to remain longer there owing to the diminished expiratory power of these portions of the lung. Carbon-dust, it is true, is found in greatest amount in the apical region in many inhalation-experiments; but, according to the views of Calmette and Guérin, the foreign matter must be first swallowed before this result occurs.

The study of the tubercular process by the naked eye in lupus shows that the disease may be extending rapidly in one direction, whilst it has become quiescent or resolved in another. In phthisis an examination of the anatomical picture proves the same fact. In the apical region, where the process has first started, repair may be in operation, and in some cases the reparative process may appear to be even

completed. As already stated, small foci may become encapsuled and ultimately calcified, the calcareous tubercular tissue being expectorated. The natural effort at repair is perhaps always in operation, though in the great majority of cases it ends in failure. The essential factor in the reparative process is the proliferation of fibroid tissue caused by the irritative nature of the bacillus or its toxin; this produces a capsule which shuts in the caseous focus. The fibroid growth may extend into the neighbouring pulmonary tissue when the disease is of an unusually chronic type, constituting the so-called *fibrous phthisis*. This sclerotic change is, moreover, often observed as a conservative process even after excavation has occurred, in which case it starts in the granulation-tissue lining the cavity, and by the slow cicatrisation or shrinking of the new fibrous growth small-sized cavities may be entirely obliterated after their caseating contents have been expectorated.

These facts explain the variations observable in the morbid anatomy of phthisis. In the same lung all the above changes may be observed at the autopsy in different portions of the organ, extending downwards from within about an inch of the apex to the lower lobe. Even in a fair-sized cavity the reparative process may be well started or established, as seen by the production of a smooth limiting membrane resembling a mucous surface, with its external or peripheral surface puckering the surrounding pulmonary tissue, whilst at some area in its wall the bacilli are actively forming tubercles, some of which show signs of caseation and necrosis as the pulmonary tissue becomes steadily invaded.

The pneumonic consolidation stage is, as might be expected, often found in one part of the lung when excavation has been advanced in another portion, and the miliary granulations of the first stage may be scattered throughout the affected areas, which become deeply pigmented as the fibrous tissue-proliferation advances, causing shrinking of the lung and involving the pleura. This latter is not often the seat of marked tubercular growths, but it is always much thickened by a chronic dry inflammation which glues the affected portions of the lung to the chest-wall by the formation of dense adhesions. These prevent the perforation of the pleural cavity by the rupture of caseating or necrosing tubercles. In the absence of adhesions, the first signs of the disease may be those caused by an escape of air into the pleural cavity; in the later stages a pyopneumothorax may suddenly develop, when a small cavity bursts and discharges its contents between the layers of the non-adherent pleura. The bronchial tubes are sometimes dilated throughout the entire lung owing to the constant pressure caused by coughing. In the affected areas the tubes may be dilated

by the traction of cicatrising bands of fibroid tissue which separate their walls.

The bronchial lymphatic glands are almost as frequently involved as the pleura; in children they perhaps, never escape. This bronchial-gland tuberculosis may be primary or secondary; in some cases the bacilli first reach the glands, whence they are distributed to the peribronchial lymphatics; whilst in other instances it would seem that the glandular infection is quite recent, being caused by invasion from the diseased foci in the lung-tissue, the lymph-current being apparently capable of flowing backward owing to blockage, as believed by Cornet. Tuberculosis of other organs exists in all very old-standing cases, the peritoneum or mesenteric glands very rarely escaping owing to the bacilli being swallowed in the saliva or sputum; and the tonsils and larynx are often affected. Amyloid degeneration of the kidney, spleen, liver, and intestinal membrane is not infrequent, especially where the invasion by streptococci and staphylococci has caused profuse suppuration in extensive cavities. The pericardium and endocardium are not rarely involved. The muscle and liver cells are usually the seat of extensive fatty degeneration, and even the nerves show signs of a degenerative change, while the membranes of both cord and brain may exhibit miliary or caseating tubercles.

Symptoms.—The *onset* varies greatly; probably its acuteness is a matter of dosage, though the varying natural resistance of the individual must play a part. Where a large dose of the bacilli is introduced through the intestinal canal, or in the inhaled air (as believed by many), or from a latent ruptured focus in some distant part of the body, the onset is acute, with all the symptoms of an attack of tubercular fever which characterise the onset of a general miliary tuberculosis. This form of pulmonary tuberculosis may have its lung-symptoms entirely overshadowed by the constitutional disturbances caused by the general infection. Acute miliary tuberculosis of the lung cannot, however, always be clinically separated from pulmonary phthisis, though the latter in its typical condition commences usually as a chronic process. Thus there is a latent form of pulmonary tuberculosis in which the first symptoms may be those of an acute miliary tuberculosis of the lung caused by the rupture of an old encapsuled caseating mass at the apex of the organ. Moreover, a large dose of the bacilli by any route may cause a general infection, ushered in with intense fever and malaise, the case afterwards passing through the typical phases of a chronic phthisis as the brunt of the invasion eventually falls upon the lung.

Cases of acute phthisis have an onset marked by pneumonic signs of such abruptness that only a pneumococcic infection may be suspected for several days, one entire lobe being

rapidly solidified. More frequently the onset is characterised by equal abruptness, but the signs are those of a diffused or scattered bronchopneumonia, which rapidly passes into caseation and excavation, constituting the well-recognised type of *phthisis florida*, in which both lungs rapidly break down. These acute varieties of phthisis differ in no way from the more common chronic form save in intensity. They are liable to supervene at any stage of the chronic type of the disease when a large focus bursts into a bronchus and infects a considerable portion of one or of both lungs; and even when such onset is sudden and severe the case may fall back into the typical chronic variety.

The onset in the common chronic form of phthisis is usually most insidious. The first symptoms of illness are frequently those due to gastric derangement, causing loss of appetite, dyspepsia, and nausea, often passing into vomiting, especially in the morning or early part of the day. Anæmia or chlorosis is generally present, the patient loses flesh, and slight evening feverishness may be detected.

In another common class of case the first symptom of the onset may appear like 'a bolt from the blue' in a copious hæmoptysis occurring in an individual apparently in perfect health. On the cessation of the hæmorrhage no pulmonary signs may be detected for a long time, though it is now accepted generally that such early hæmorrhage should be interpreted as the result of otherwise latent tubercle, and not as the cause of it—a view formerly accepted without question.

The most common of all the varieties of onset is the bronchitic. The patient suffers from cough, which he supposes to be that caused by an ordinary catarrh; it does not pass off, however. Not infrequently he becomes so accustomed to it that he is unconscious of coughing till the anxiety of his relatives compels him to seek the advice of a physician, who may not even then be able to detect any other evidence of the disease.

Pleuritic pain caused by a patch of dry exudation upon the pulmonary pleura rubbing against the parietal layer in deep breathing, sneezing, or coughing, is often the first symptom to arrest suspicion. In this connection a very important matter must be considered. Pulmonary phthisis so frequently supervenes in individuals who have previously suffered from an attack of simple pleurisy with effusion, long after recovery from the latter, that there is good reason to believe that the pleurisy is the cause of the subsequent phthisis. The attack of inflammation leaves the pleural and pulmonary tissues in a condition of diminished resisting power, which determines the advent of phthisis in the same manner as trauma does. This is quite a different matter from the local pleurisy which often heralds the advent of

phthisis, as in this latter case the pleuritis is the direct result of the local tubercular deposit near to the lung-surface.

Laryngeal symptoms are sometimes the first evidence of any departure from the healthy standard, as observed in hoarseness and muffling of the voice. Here again the interpretation of the onset is beset by the same difficulty: Is the voice-change the result of a secondary laryngeal tuberculosis which has supervened upon a latent pulmonary affection, or is it but the local manifestation of a general catarrhal condition? It is perhaps safer to regard it from the same point of view as the prodromal hæmoptysis, and consider it as evidence of the disease having already made some progress in a latent manner, but it certainly is not correct to always regard it as proof that the tubercle has already invaded the larynx. It may be the result of recurrent laryngeal nerve- paresis, caused by the pressure of tubercular bronchial glands or pleural thickening and adhesions whose presence affords no other indication of mischief.

It is manifestly difficult to give a picture of a disease so very variable in its symptomatology, progress, and duration. Some cases terminate fatally in a few months after the first signs have become obvious, whilst others will drag on an existence for perhaps seven to twelve years. The patient, after passing through the prodromal period, in which the symptoms of the bronchitic, pleuritic, dyspeptic, anæmic, laryngeal, or hæmorrhagic onset have been observed, is discovered to be phthisical. The usual or most common course for the disease to pursue is something like the following: Cough becomes prominent, muco-purulent expectoration appears, there is some chest-pain and shortness of breath, and sometimes blood is observed in the sputum—symptoms which are obviously of local origin, pointing to the implication of the lung. These are marked by signs of a more or less definite character which enable the physician often to divide the progress of the disease into the three stages commonly spoken of as those of (1) *consolidation* or tubercular infiltration, (2) *softening*, and (3) *excavation*—though all these stages may be sometimes observed in different portions of the same lung at the same time.

By the termination of, say, from four to six months, with the deepening of the above symptoms, others are added on, which are indicative of a constitutional reaction. Fever, which is usually slight at first, now becomes of a markedly hectic type, in which sweating is a prominent feature; emaciation, present from the beginning, steadily increases, and before the termination of another six or nine months the patient's aspect is one of marked wasting, with muscular debility, often intensified at intervals by exhausting diarrhœa and profuse purulent expectoration, so that usually before

the termination of a couple of years from the onset he sinks slowly into his grave, unless life be suddenly terminated by a profuse hæmoptysis, pneumonia, or other complication.

In the acute variety of phthisis, recognised as galloping consumption or phthisis florida, the disease runs through the above-mentioned phases often within a period of six to twelve weeks. In the so-called fibroid phthisis the duration may be protracted from the initial hæmoptysis of the onset for a period of a quarter of a century before the final termination.

The above symptoms and signs will be discussed *seriatim*.

Cough.—This is the most constant and it frequently is the earliest of all the symptoms. It is a reflex phenomenon caused by the irritation of the chemical products excreted by the bacillus; often at first referred to the pharynx or larynx, it is complained of as a continual tickling, which may compel not only involuntary paroxysmal explosive efforts to dislodge a supposed foreign body, but may induce the patient to perform voluntary hawking exercises to clear his throat. It is of a dry and hacking character, being at first usually unaccompanied by any expectoration, and for a considerable period it may be experienced only in the mornings, in which case it very commonly eventuates in an attack of vomiting. In irritable neurotic individuals the early cough of phthisis is always a prominent symptom; apparently it becomes a habit-cough, the patient often being unaware of or oblivious to its frequency.

As softening of the tubercular foci occurs and perforation of a bronchiole takes place, the dry hacking character of the cough changes. It tends to become paroxysmal, in severe kinks, as the highly irritating necrosed tubercular masses come into direct contact with the sensitive bronchial and tracheal membrane, producing more or less bronchitis with secretion. It may then present for a long period the characteristics of a loose bronchial cough, with muco-purulent secretion brought up at regular or irregular intervals. Later on, as cavities form, the cough-characteristics may be quite distinctive, the patient obtaining more or less complete rest and freedom from coughing for considerable periods after a paroxysm has enabled him to pump out a large cavity. These paroxysms occur after a period of bodily quietude or after sleep, during which the cavities have been slowly filling with the purulent secretion; hence they are usually constantly present in the mornings, though at any period of the day or night a sudden change of position may bring on an attack. When laryngeal mischief supervenes, a croupy character is added to the usual loose-sounding cough, and the advent of pleuritis is often first detected by the severe stabbing pain which is brought into evidence during coughing. It must

be remembered that some cases of phthisis run through a considerable part of their course without cough, though there is a liability to error in estimating the frequency of this peculiarity. The writer has been often surprised during the examination of a chest, in which marked softening of the lung was evident, by the patient affirming that he had no cough, whilst slight coughing was going on all the time, the patient himself being quite unconscious of the effort.

In children the cough may be observed to be modified or to be altogether caused by nerve-pressure produced by masses of tubercular lymphatic bronchial glands. In hysterical women the early, and indeed the later, cough of phthisis may have engrafted upon it all the characteristics peculiar to the barking cough and aphonic phenomena of this neurosis.

Chest-pain.—As already stated, this may be the first symptom and the only one during the prodromal period. The pain is usually confined to the region of the apex, and may be interpreted as the result of a localised dry pleuritis over a latent superficial area of pulmonary tubercle. It is intensified by coughing and sneezing, and is nearly always confined to the affected side. There is generally considerable hyperæsthesia of the skin over the seat of pain—*i.e.* about an inch below the middle of the clavicle. This early stabbing pain is different from the muscular and neuralgic pains which commonly arise later on in the disease, and which may be felt near the base of the thorax or along a particular intercostal nerve or about the angle of the scapula. Like cough, the chest-pain may be absent throughout the entire course of phthisis.

Expectoration.—In the typical condition this is absent at first, the cough being a dry one, though repeated hawking may bring up small pellets of mucus and plugs from the tonsils. Later on mucus of a clear boiled-sago appearance is brought up; this gradually becomes more or less tinged with a greenish hue as pus is formed, and by the time that softening is in progress the muco-purulent expectoration presents all the characteristics of the typical secretion from a chronic bronchitis. At this stage repeated inspection may reveal small greenish or grey particles in some specimens, which can be demonstrated to consist of cascating foci which have been discharged into a bronchus. After the formation of cavities the expectoration becomes more profuse and purulent, and presents the nummular appearance, each sputum being separate and airless and somewhat coin-shaped when dropped into water, and on close inspection a film or coating of clear viscid mucus may be distinguished around its margin. In large freely secreting cavities the pus may be so abundant that the sputa run into each other and form a greenish, creamy,

liquid emulsion, in which all traces of the nummular character are lost.

The separate masses, being devoid of air, readily sink to the bottom of a vessel containing water, and they retain their circular form for a considerable time ; but other air-containing and more purely mucoid sputa are also expectorated from time to time, and may be seen floating upon the surface of the water in the spittoon. If blood be present, it may be visible in specks or streaks or intimately mixed with the pus, or it may give to it the brick-dust, prune-juice, or tobacco-spit appearance. Various colour-producing bacteria may simulate the characteristics of blood, and when *B. pyocyaneus* is flourishing in the cavities the green colour of the expectoration is always highly marked.

The quantity of expectoration varies enormously in different cases, and in the same patient it fluctuates widely at different times. It is quite common to find the chest-signs well marked and indicative of great activity without any expectoration ; in these cases the process is very probably of the peribronchial or perivascular type. Children generally swallow their sputa, and some adults unconsciously or involuntarily do likewise ; but such patients betray the presence of copious secretion by the loose character of the cough. When the reparative process gets the upper hand in the struggle, the expectoration gradually diminishes as the cavities shrink by cicatrisation ; whilst in ordinary incurable cases the amount steadily increases, or is at times augmented by the supervention of the repeated attacks of general bronchitis to which phthisical patients are especially prone. During these exacerbations the sputum may for the time lose its nummular characteristics ; and for another reason this feature of the individual sputa should not be relied upon—viz. it is practically identical in appearance with the sputum of chronic non-tubercular bronchiectasis, and can only be differentiated by microscopical examination.

The main point in the investigation of early phthisis is the discovery of the tubercle bacillus. This can only be demonstrated after careful staining of a small portion of the sputum. Various stains are employed for the purpose, the most convenient and reliable being that of Ziehl, which consists of a solution of fuchsin in alcohol and carbolic acid ; this staining medium maintains its activity unimpaired for indefinite periods. A small quantity of the most suspicious particles selected from a specimen of sputum is smeared over a cover-glass ; this is slowly passed a few times over a spirit-lamp flame, after which a large drop of the solution is applied and allowed to steam or simmer gently, but not to boil, for less than a minute in the flame. The excess of staining material being washed away by a stream of water, the remainder is discharged by agitation in a 25 per cent.

solution of sulphuric acid. The cover-glass having been next immersed in absolute alcohol and washed in water, a solution of methylene-blue is applied for about half a minute ; after washing the latter away, the specimen, upon being dried, is mounted in a drop of oil and examined by an oil-immersion lens capable of magnifying 800 diameters. The bacilli retain the primary stain, while all the other micro-organisms and tissue-elements are decolorised by the acid and afterwards take the methylene-blue dye.

The bacillus of tubercle is a rod-shaped slightly curved and sometimes beaded organism of about one-third of the diameter of a red blood-cell in length ; it is recognised at once in a specimen prepared in the above manner by its red stain in the midst of the blue. Some ovoid areas may be observed in the course of the rod which do not stain as well as the remaining portions of the organism, and these have been conjectured to be spores ; but nothing is definitely known regarding the sporulation of the tubercle bacillus. The only organisms likely to be mistaken for the specific bacilli are the bacillus of leprosy and the smegma bacillus. The other micro-organisms, fungi, sarcinæ, &c., found in the sputum are of but secondary importance. Cells from the epithelium of the different parts of the air-passages and leucocytes are found in abundance, and often red blood-discs are present. Melanin and pigment-cells are also constantly found, and after boiling the sputum with liquor potassæ yellow elastic fibres may be commonly detected, which prove that destruction of the pulmonary tissue is taking place. The specific bacilli may exist in enormous numbers, as in a pure culture, but sometimes only a few may be found in the field at once, and occasionally they may be absent when the caseating focus is not in communication with a bronchus. Their presence is absolute proof of the tubercular nature of the pulmonary disease, and they are certain to be demonstrable by repeated examination. The great value of sputum-staining lies in the fact that often the bacilli can be detected before any pulmonary *signs* develop.

Calcareous particles may sometimes be observed by the naked eye in the sputum of phthisical patients whose tubercles have undergone partial resolution, and the trained eye can frequently detect small masses of elastic tissue which can readily be verified by the use of a pocket-microscope without resorting to boiling.

Chemical examination of the sputum yields results as yet of but slight clinical value. Müller finds that whilst the sputum of bronchiectasis will liquefy gelatin, the expectoration from phthisical cavities will not do so. The heavy, sweet, and mawkish odour of typical phthisical sputum is often replaced by a disgusting fœtor when the discharge of

the cavities is mechanically interfered with ; it has little clinical signification, save that it is evidence that the other micro-organisms are flourishing.

Hæmoptysis.—If all cases of phthisis be taken together, and their entire course studied, it would be probably found that hæmoptysis is, upon the whole, more frequently met with as one of the very earliest symptoms than as a sign of the late stages of excavation. Already, in speaking of the onset of the disease, the cause of its early appearance has been discussed ; it is now universally accepted as being, even in these early cases, the result of a necrotic process in the tubercular growth which has already invaded the coats of a small vessel, when this latter bursts at its weakest spot under the pressure of the circulation.

The reason of its comparatively rare appearance in the later stages of the disease, when the pulmonary tissue is being extensively excavated and when copious hæmorrhages might be expected to be the rule, is explained by a study of the morbid anatomy of the disease. The irritation produced by the toxins of the bacillus tends to cause obliteration of the lumen of the blood-vessels in the neighbouring areas of the lung. Hence, these being usually found impervious, their subsequent involvement by the necrotic process does not eventuate in hæmorrhage. Cornet points out that in the very early stages of the disease the proteins are still scarce and the obliteration does not occur.

These facts explain the differences between the early and late hæmoptysis ; in the former the amount of blood lost is commonly small and may resemble a capillary oozing, whilst the bleeding in the cavernous stage, which, as already seen, usually comes from aneurismal dilatations on the arterial wall, may in rare cases be so copious as to cause speedy death. The immediate exciting cause of the hæmorrhage in either case may be a fit of coughing, but more frequently it appears suddenly, independently of cough, welling up the trachea into the mouth and exciting a sensation as if some saline substance was in contact with the tongue. Very commonly, however, slight streaks or punctate spots of blood have been previously at some time observed in the sputum. The blood is usually bright-red, mixed with air-bubbles and often with muco-purulent expectoration, which at once betrays its pulmonary origin ; but sometimes, especially during the night, the blood upon reaching the pharynx is swallowed during sleep, and may be ejected in the morning by an act of vomiting, in which case the explanation of the secondary hæmatemesis may be overlooked. Rarely a large hæmorrhage may fill an extensive cavern in the lung and cause sudden collapse without external signs of bleeding.

In some phthisical patients the tendency to recurrent hæmorrhages is marked all through the different stages of the disease, and these have been regarded as constituting a type of phthisis known as the '*Hæmorrhagic*,' to be distinguished, however, from that formerly supposed to be produced by bleeding, and which was styled by Niemeyer '*Phthisis ab Hæmoptoe*.'

The first-mentioned hæmorrhagic type is not, however, the result of any peculiarity or variations in the bacillus, but probably depends upon some blood-condition or congenital weakness in the vascular walls akin to that observed in bleeders. Hæmoptysis sometimes makes its appearance in phthisical women at the menstrual periods, and it is a noteworthy fact that it is extremely rare in young phthisical children. The occurrence of early hæmorrhages is a serious problem in diagnosis and prognosis, as very frequently there are no other signs present, and physical examination of the chest gives negative results. Every possible source of the bleeding should be carefully eliminated by an examination of the nose, pharynx, gums, mouth, and larynx, and by a scrutiny of the conditions preceding the hæmoptysis. Thus, as regards this latter, blows on the chest and violent muscular exercises, the presence of valvular lesions, especially that of mitral stenosis, aortic aneurisms, gastric ulcer, purpura, hæmophilia, &c., must be carefully considered. Due weight must be given to the fact that some injury or violent exertion occasionally is the immediate cause of the rupture of the vessel already seized upon by the latent tubercular process. The problem is rendered more difficult by another fact too often not recognised—viz. that the fresh blood lying in the small tubes causes râles which for a time simulate phthisical signs at a distance from the real site of the latent mischief in the apex. It must be always borne in mind that a considerable percentage of individuals who have been attacked by hæmorrhage from the lung never develop phthisis, and this percentage rises greatly in the group of cases where violent exercises, as running, swimming, rowing, singing, &c., had preceded the hæmoptysis, and when the subjects showed no family-history of tuberculosis and had been in previous robust health. It is unnecessary to point out the great importance of a microscopic examination of the blood in all cases, though a negative result is of little importance. Professor Lindsay lays stress upon a feature in the early hæmoptysis of phthisis—i.e. it does not cease suddenly; and this, he considers, constitutes a diagnostic point of some importance.

Laryngeal Symptoms.—These have been dealt with in describing the different varieties of onset in chronic phthisis, since many cases show the first departure from health in some abnormal voice-change, as hoarseness, muffling of the voice,

weakness, or total aphonia. At a later stage the laryngeal symptoms are nearly always caused by a secondary involvement of the larynx by the tubercle bacillus. Occasionally, however, the voice-changes are, even when appearing late in the disease, due to paralysis, the result of pressure on the laryngeal nerve-trunks by enlarged bronchial lymphatic glands. The same explanation may be applied to the *dysphagia* which sometimes is present even when no tubercle has invaded either larynx or pharynx. The writer thinks that the early laryngeal symptoms, which are sometimes very marked but which pass away as the chest-signs develop, may be due to an invasion of the bacilli, which is repulsed or eventually overcome by the natural resistance of the laryngeal mucous membrane; this is rendered likely by the fact that the laryngoscopic signs in these early cases are so marked that the diagnosis of tubercle is sometimes made by the throat-specialist in cases which afterwards turn out to be pulmonary phthisis, but where laryngeal ulceration never develops.

Dyspnœa is often observed in phthisis. Commonly it is a late symptom, but it may be in rare cases the first evidence of the disease when a small necrotic area on the surface of the lung bursts into the pleural cavity, causing extreme dyspnœa, the result of a rapidly developed pneumothorax; and such an event is usually induced by some violent effort.

Unless when appearing during exercise, dyspnœa is not often due to the mere loss of lung-substance, since a very remarkable degree of excavation, even in both lungs, may be accompanied by quiet breathing. The advent of breathlessness in phthisis, as in pleuritis with effusion, is more a matter of the degree of rapidity with which the breathing-space is obliterated, since it is often a very obvious symptom in an acute miliary tuberculosis, and in a chronic phthisis when pneumonia, bronchopneumonia, or pleurisy suddenly supervenes. It has been supposed to be in some cases caused by irritation of the vagus terminals or pressure of enlarged glands on the trunk of the nerve, and it may become marked when a cavity fills, and cannot be emptied owing to the weakness of the expulsive mechanism in the late stages of the disease. Like cough, pain, hoarseness, expectoration, hæmoptysis, or any other local symptom, dyspnœa may be absent throughout the disease. It is much more likely to be present when emphysema is marked, and it is certain to be a prominent feature when the phthisis supervenes or is associated with asthma or heart-disease, especially mitral stenosis.

Of the constitutional symptoms of chronic phthisis, the most important, from the view of diagnosis and prognosis, is the rise in body-temperature.

Temperature.—A certain degree of pyrexia is practically the invariable rule in phthisis; even in the most chronic fibroid form, which is apparently apyrexial, frequent registration of the temperature will always reveal an elevation beyond the normal at times or during longer or shorter phases in its slow course. The cause of the fever is undoubtedly the absorption of the toxins manufactured by the bacillus of Koch, and at a later stage, when the septic products of streptococci and staphylococci are absorbed from the walls of cavities secondarily infected by these pus-forming organisms, the temperature reaches a further height as the result of the mixed infection. It is thus obvious that the temperature-chart supplies information which may be interpreted as evidence of the extent of the tubercular deposit, and in some degree also of the activity of the destructive process in the lung.

The registration of the temperature affords often the only indication of the disease in the early stage before the appearance of any signs in the lung; but to be of any value in these cases a four- or six-hour chart must be diligently kept for some time. The most common type of the temperature at this stage is the *intermittent*; the thermometer registers a slight rise (100°) in the early evenings, and the morning temperature is normal or sub-normal.

Two points in connection with the early pyrexia of phthisis are noteworthy. The pulse-rate is nearly always accelerated much more than the degree of fever warrants, and owing to the effects of even small doses of the tubercle-toxins the thermic centres become hyper-sensitive. This latter feature is of great value in diagnosis, and Schüle and Penzoldt place the chief reliance upon it in the elimination of other febrile states. It is demonstrated by testing the temperature, preferably in the rectum, before and after some muscular exercise, when the elevation usually manifests itself even when otherwise no trace of pyrexia may be appreciable. Mental exercises, and even slight emotional disturbances, may likewise be found to leave their impression upon the temperature-chart. Though these changes also occur to a slight degree in health, the phthisical elevation is invariably of longer duration.

At a later stage, when softening or excavation is progressing, the type generally changes to the *remittent*, in which the temperature is constantly above the normal, and the evening rise usually exceeds $1\frac{1}{2}$ or 2° F. A *continuous* type of fever is sometimes met with in which the temperature is always above the normal, but the evening exacerbation does not exceed the morning pyrexia by more than about a degree and a-half; this type is commonly seen for considerable periods in phthisis florida, or galloping phthisis, in which variety of the disease the evening rise very frequently keeps

at a level as high as 104° . It is frequently also caused in chronic cases by the supervention of pneumonia.

The pyrexia of phthisis, however, like the other symptoms and signs, is most variable and mixed, all sorts of curves being observed sometimes in the same case at different phases of its progress; but this very tendency to variation constitutes its main distinguishing feature when contrasted with typhoid, malarial, and other fevers. In the severe hectic fever of phthisis, which is usually accompanied by much sweating, it is not an uncommon event to observe a sudden drop from 105° to 97° at any period of the day or night. Sometimes a hyperpyretic temperature (107°) is rapidly followed by a collapse-temperature (95°), especially in the very advanced stage of the affection, when septic organisms are rapidly multiplying in the cavities. When it is remembered that in the lung miliary tubercles are forming in one area and pushing out into the healthy pulmonary tissue, whilst at the same time large cavities are being excavated in another area and extensive tracts are undergoing pneumonic or bronchopneumonic consolidation, it is easy to conceive how varying and complicated from time to time may be the curves and angles on the temperature-chart. Hence any attempt at a classification of the pyrexia of phthisis is of little clinical value, though the general survey of the chart affords the most valuable and reliable guide to the activity of the disease. Sometimes the ordinary characteristic of a higher evening range is reversed, the afternoon temperature being for days always normal or much lower than the morning register, constituting the *inverted* type often seen in children, and not rarely observed in miliary tuberculosis.

A drop to the normal temperature, which may be maintained for weeks or even months, is not a rare event, and indicates the evacuation of the bacilli and their irritating toxins; it may usually be accepted as a good omen and as evidence that the reparative processes are in active operation.

Chills or *rigors* often precede the rapid rises of temperature, and sometimes these closely simulate malarial attacks, especially when followed by drenching perspiration after the fall.

Sweating.—Excessive perspirations may be an early sign, but much more frequently they only become marked in the softening and excavation stages. At first the sweating is most obvious at night, the patient awaking with the hair of the head quite wet; but later on in the disease the sweating may be of the most profuse degree at any hour of the day or night. Very often it is closely associated with the fever, both being produced by the same cause—viz. the absorption of tubercular and other toxins; and its profuseness depends upon the previous height of the temperature, or, rather, upon

the range of its sudden variations, being often drenching when the collapse-temperatures occur. Like the other signs and symptoms, sweating may be absent throughout or it may occur independently of pyrexia, especially in weak neurotic girls. In all cases the sweatings are accompanied or followed by a sense of severe exhaustion.

Pulse-rate.—This is as a rule markedly accelerated; in the early stages, owing to the rapidity of the pulse being out of proportion to the degree of fever, this feature, as already mentioned, possesses a certain degree of diagnostic significance. Even when there is no fever in the case, the toxins exercise a paralytic action upon the vagus-centre, so that tachycardia becomes marked after but slight exertion. All through the progress of the disease, however, the pulse-rate is high, especially when the pyrexia is constant; and the rate may keep high after the fever falls, though the breathing remains quiet and natural. Tachycardia as marked as that observed in exophthalmic goitre may occur from the pressure of enlarged glands on the nerve paralysing the inhibitory fibres. The pulse of phthisis, in addition to its rapidity, is usually small, soft, and weak, owing to the reduced blood-pressure and the cardiac debility always present.

Emaciation.—This may be an early symptom; as a rule, it is in direct proportion to the amount of fever, though not necessarily the direct result of the pyrexia. It is obviously intensified by the presence of gastric disturbance, loss of appetite, and vomiting, and is always accentuated by intestinal implication, which produces diarrhœa. In the general wasting the fat disappears first, but ultimately every tissue in the body, save in some cases the hepatic, suffers; and in the late stages of the disease the muscular atrophy becomes extreme, the patient resembling a living skeleton. Arrest of the disease is shown by an improvement in the body-weight; but though this increase is always a favourable omen, too much importance is not to be attached to it. Nothing is more remarkable in the study of phthisis than the obese condition which is sometimes observed in sanatorium-patients, whose weight has been increased by amounts of even two and three stones, though the physical signs in the lung remain in no way improved. Nevertheless, the increase in weight must be regarded as evidence of a diminution in the production of the toxins, since these, independently of the fever which they cause, certainly tend to induce marked emaciation. That the emaciation is not alone caused by the fever is evident in a small proportion of cases where marked wasting continues progressively through a long apyrexial period, in which the patient's appetite may remain good.

Muscular Weakness is often marked even in the early stage, before wasting of the muscles or fever is sufficiently obvious

to attract attention; this, along with some anæmia and rapidity of pulse, constitutes a very suspicious combination of symptoms when no other explanation can be assigned. Often mental lassitude and despondency are present, but the late stage of the disease is often characterised by a remarkable hopefulness, which may continue till a few hours before death.

Cutaneous signs are, for the most part, inconspicuous, if we except the branny desquamative areas of the skin in which the pityriasis versicolor fungus (*microsporon furfur*) flourishes. In the late stages pityriasis tabescentium appears, with the extreme marasmus as the skin atrophies and the superficial dry cells are constantly shed. Often much pigmentation of the skin is observed and the cutaneous surface is for a time greasy, but more commonly the skin gets dry, pale yellow, and wrinkled. The hair of the head, though sometimes luxuriant and glossy, as a rule becomes dry, brittle, and sparse. A very valuable sign in early phthisis, in the experience of the writer, is the frequent occurrence of a profuse crop of fine, long, satiny, curved hairs below the nucha, extending downwards to the suprascapular fossæ. It cannot, however, be interpreted as the result of the disease, but it certainly seems to indicate a congenital predisposition to tuberculosis.

The finger-tips become clubbed and the nails incurved as in cardiac disease occurring in early life. Though this drum-stick enlargement is only caused by some hypertrophic change in the soft tissues over the last phalanges, sometimes the rare and highly interesting condition known as *hypertrophic pulmonary osteo-arthritis* is observed, in which the terminal phalanges of the fingers, toes, and other bones become the seat of a genuine hypertrophy.

The early hectic red spot or blush over the malar prominences often gives an obvious characteristic appearance to young and pale phthisical subjects. Many observers attach great value to the frequency with which the gums are observed to be deeply reddened in the early stages of the disease. Of more importance, in the writer's experience, is the frequency of the presence of extensive necrosis of the teeth, to which the redness of the gum-margin is usually due.

Edema often accompanies the early anæmia or chlorosis, but it is slight, and is only to be detected when the patient has been long sitting up or moving about; in the later stages general anasarca is present, as renal changes or amyloid degeneration, with considerable albuminuria, set in.

Signs of great increase in the irritability of the superficial chest-muscles is nearly always present in advanced cases, a slight tap over the pectoralis being followed by a small tumour or elevation of the wasted muscular fibres, which may remain for several seconds. This phenomenon is, however, sometimes, though rarely, witnessed in other wasting diseases.

Blood-changes.—These are not of much clinical importance, and the bacillus is very seldom found in the circulating fluid. The anæmia of the early stage, it is worth remembering, is often associated with a considerable diminution in the number of red cells; but as the disease progresses there appears to be some diminution in the amount of the blood-serum, since the number of red discs usually reaches the normal five millions in a cubic millimetre of blood. Marked leucocytosis never occurs till the excavation-stage, when septic absorption is active. The serum of tuberculous blood has often been found to cause clumping or agglutination of the specific bacilli when these are added to it in beef-broth cultures; but the phenomenon is of little real value in diagnosis.

Gastric symptoms have been incidentally referred to; they may be the earliest indications of the disease, as described in the modes of onset of phthisis, and it is often noticed that the early dyspepsia, vomiting, and anorexia of phthisis pass away as the disease progresses. This is possibly due to the gastric mucous membrane becoming more tolerant of the toxins circulating in its nutrient fluid—a condition of affairs observed sometimes in chronic uræmia. When the gastric mucosa remains irritable the most serious prognosis may be safely made, as the patient continues unable to avail himself of the advantages which over-feeding supplies in the struggle against the inroads of the bacilli in the pulmonary tissue. More commonly, however, the gastric disturbances, like the fever, expectoration, and sweating, vary for considerable periods at a time, probably on account of the dosage or activity in the absorption of the toxins. Smart gastric catarrh is common in children from swallowing the sputum.

Intestinal symptoms show themselves by a diarrhœa which often alternates with constipation. The obstinate diarrhœa of the late stages of very chronic phthisis is more frequently due to amyloid disease than to tubercular lesions in the mucous membrane. Children suffer most frequently because of their habit of swallowing the sputum, which acts as an irritant; and Cornet has drawn attention to the frequency with which diarrhœa supervened during the practice of large hypodermic injections of tuberculin—a fact which proves that the toxins circulating in the blood may cause diarrhœa either by increasing secretion, by stimulating peristalsis, or by diminishing the rate of absorption of the intestinal secretion. The fact has already been emphasised that the bacilli readily penetrate the intestinal epithelium, enter the chyloferous vessels, and reach the mesenteric glands without causing any discoverable lesion or breach of surface—a fact which explains the comparative infrequency of tubercular ulceration of the bowel.

Physical Signs.—The symptoms are of less importance in the recognition of phthisis than the signs observable in the chest.

INSPECTION may reveal nothing in the early stage of the disease, and the physician in doubtful cases should be upon his guard not to attach too much importance to the type of the patient's thorax. The popular notion that almost any deformity or malformation of the chest is to be taken as evidence of a predisposition to phthisis is an error. The narrow elongated chest of individuals believed to be prone to phthisis is maintained by Colbeck not to have its sterno-vertebral diameter diminished, but to have its vertical measurements after the proportions seen in the infantile type of thorax. The scapulæ stand out, giving the chest a winged appearance; the shoulders are often rounded, and the intercostal spaces flat and wide. Whilst an individual with this type of so-called 'paralytic thorax' has certainly diminished inspiratory power, its presence should never lead the physician to diagnose the disease unless other signs are clear. It is of more importance to remember that phthisis is far more frequently met with in the most robust and symmetrical types of chest.

Inspection at a comparatively early stage of caseation will often reveal diminished mobility in one apical region, which does not expand in quiet breathing, though on deep inspiration this may not be observable owing to the elevation of the shoulders. This deficiency in expansion may be corroborated by palpation. In a later stage distinct retraction or flattening below either clavicle or at any spot in the chest-wall may be noticeable after the softening has produced excavation. The sternal end of one clavicle often appears more prominent, usually on the sound side. In advanced apical disease the lung is drawn up by the shrinking of a large cavity, so that its margins cease to cover the heart, which thus comes into direct contact with a larger area of the chest-wall, as seen in the augmentation of the apex-beat.

PALPATION usually only serves to corroborate points observed in inspection, and not rarely a diminished apical expansion is more obvious to touch than to sight. Vocal fremitus is determined at this stage of the examination; in the consolidation-stage of phthisis the fremitus is usually increased; but it must be remembered that the vocal fremitus in health is greater on the right side even as high as the apex.

MENSURATION seldom affords any information, save in cases of great retraction or where pleuritis with effusion has supervened.

SKIAGRAPHY OR RADIOSCOPY.—The X-rays afford valuable information in many cases, not so much in clearing up a doubtful diagnosis as in more accurately determining the extent of the disease. Green and Brook have shown that an early

sign is the limitation of the movements of the diaphragm on the affected side; and this is clearly demonstrated by the Röntgen rays. They were able to locate deposits in the absence of all physical signs in cases which afterwards presented marked signs. In the normal lung no shadows appear, but in dense tubercular infiltrations and in calcareous deposits in old tubercle the shadows may usually be made out without difficulty; but special skill and experience are necessary for the correct interpretation of all skiagrams of the lungs.

PERCUSSION.—Only when the area of consolidation is larger than a square inch, and near to the surface of the lung, can a distinct dulling of the percussion-note be expected, unless in children with extremely thin parietes. Large foci situated deep in the pulmonary tissue usually cause no shortening or flattening of the normal resonance, though occasionally a dull note may be brought out by heavy percussion in a suspected area which yielded normal resonance to a light stroke. The corresponding region on the opposite side must be always contrasted.

Hyper-resonance may be due to compensatory emphysema, and occasionally very careful percussion may demonstrate the presence of this change about the margin of the consolidated area. Tympanitic resonance with a high-pitched note, sometimes almost of musical quality, is usually indicative of a cavity. Percussion of the chest-wall over such an area gives a higher note if the patient's mouth be kept open; and if he be made to take a deep inspiration this latter manœuvre may raise the pitch so high that no tympanitic note results owing to the tension of the chest-wall. The note is lower over large cavities, especially those with small orifices. The *cracked-pot sound*, or *bruit de pot fêlé*, may be often elicited by percussing the chest-wall over a superficial cavity when the patient's mouth is kept open and the physician strikes with a rapid and short stroke. This sound is sometimes heard on percussing the thin chests of children who are crying or singing. It resembles the sound produced by entwining the fingers of both hands with their palms almost in contact and then striking the back of one hand against the knee. The ideal condition for its production in the phthisical chest is a cavity below the clavicle with a small orifice leading into an open bronchus.

An amphoric or bell-like quality often accompanies the increased or tympanitic resonance over a cavity when its walls are firm and its lining smooth; it may also be obvious when tympanitic resonance is absent.

Owing to the frequency of the tubercular deposits being located in the apex, slight shades of dullness in this region are of considerable importance in the early stages, but the absence of apical dullness may be obvious when the foci are numerous,

with islets of healthy lung between. The diminution of resonance must be interpreted as simply implying that the lung is consolidated and airless; it is practically identical with the phenomenon elicited by percussing a pneumonic lung, and is only suggestive of tubercle by the smallness of its extent and by its apical site.

AUSCULTATION.—The ear should first search for the normal vesicular murmur, noting its presence or absence and any modifications which it has undergone; afterwards adventitious sounds must be closely examined. The apical regions are obviously the most important in the early diagnosis of phthisis, but it must be remembered that miliary tuberculosis may invade the lungs from apex to base and give no auscultatory evidence of its presence till secondary bronchial or pneumonic changes are set up. It is true in the main, therefore, to say that the first signs of chronic phthisis are those of either a localised bronchitis or pneumonia, or of both these conditions combined; but the importance of the signs lies only in their circumscribed distribution and in their apical situation.

The normal vesicular murmur is audible usually only in inspiration during natural quiet breathing, the expiratory sound being so feeble and short in duration as to be scarcely audible. One of the earliest changes in the tubercular process is the intensification and prolongation of the expiratory portion of the breath-sound. It comes to resemble the sound heard in the normal lung when the stethoscope is placed over the interscapular region where the bronchi bifurcate, or when the physician listens to the breathing in the vicinity of the trachea or larynx. It is therefore usually styled *bronchial breathing*, and only differs in degree from the harsher and more intense blowing sound heard over the consolidation of lobar pneumonia. The main feature, therefore, in this early modification of the vesicular murmur is the harshness, roughness, intensity, and increased length of the expiratory sound, which may be described at first as *broncho-vesicular*, but as the bronchitic and pneumonic process extends the vesicular breezy character of both sounds is entirely lost, and the breathing becomes typically bronchial and blowing.

In many cases of early phthisis this blowing breathing at one apex is absent, and instead of it the only modification of the normal sounds is *feebleness*. This may be so marked, owing to the obstruction of the air to the bronchioles and alveoli, that the vesicular murmur of inspiration may be entirely inaudible, except in forced respiration.

Cog-wheel Respiration is often an early sign of much value. This phenomenon consists in a modification of the rhythm of the natural breath-sounds, which become interrupted, jerky, or wavy, owing to some obstruction in the bronchi.

It is usually marked when the pleura over the tubercular nodules is involved, and it is often associated with some pain in deep inspiration. Like the bronchial breathing just described, it is of no significance unless when circumscribed or confined to the apical region, as it is often heard in examination of the chests of nervous children.

Changeable or *Metamorphosing Breathing* is the usual name given to modifications which are sometimes heard in bronchial respiration. The inspiratory sound is harsh at its commencement and becomes hollow as it passes into the expiratory sound, which from its commencement is soft and low-pitched in contradistinction to the inspiratory. To this type of breathing the term *metamorphosivendes Athmen* is applied by Seitz, and it is to be practically accepted as evidence of a cavity. It must also be remembered that typical bronchial breathing is often heard over superficial firm-walled cavities communicating with large bronchi.

Amphoric Breathing is the name given to the bell-like modification of the respiratory sound which confers upon it a metallic quality; it is heard over large smooth-walled cavities, and is very marked in open pneumothorax. These latter modifications of the normal breath-sounds are usually associated with râles and other adventitious sounds.

The auscultatory changes in the breath-sounds over an area in which the tubercular process has proceeded to cicatrization are various. Often there is no modification of the vesicular murmur to be detected, save feebleness of the sounds due mainly to the pleural thickening and fixation of the apex, in which case there will be also marked dulness and flattening; but more frequently there are permanent prolongation and intensification of the expiratory sound, which remains bronchial in character.

Adventitious Sounds.—In addition to the above-mentioned modifications of the normal sounds, all the râles, rhonchi, and crepitations heard in bronchitis and pneumonia may from time to time be present. Their significance therefore depends, like the other signs of phthisis, not so much upon any specific or pathognomonic characteristics, but mainly upon their site and localised distribution. An important indication of excavation is, however, manifested by the supervention of metallic or amphoric qualities upon râles and rhonchi, which then often partake of a distinctly gurgling character.

The adventitious sounds of phthisis are usually classified under the heads of *dry* and *moist* rhonchi or râles; but since different writers apply the same name to different kinds of râles the description of the auscultatory phenomena of phthisis is generally confusing. It is well therefore to briefly define the sense in which these terms are used, avoiding

debatable points in the explanation of the mechanism of their production as far as possible. There is one sound which when once heard can never be mistaken: this is the so-called fine crepitant râle characteristic of the early stage of lobar pneumonia, and only heard during the inspiratory act. It can be exactly reproduced by rubbing a lock of hair close to the ear between the finger and thumb; the phenomenon is very commonly referred to as *fine crepitation*. It is believed to be produced in the alveoli and smallest bronchioles by the separation of their walls when these are coated over with viscid secretion. It is limited almost invariably to inspiration and is not heard till the inspiratory sound has been well started, a number of fine crackles or minute explosions occupying the last half or two-thirds of the inspiratory sound.

This fine crepitation is often heard about the margins of actively spreading tubercular foci, and though usually classified as a moist sound it does not convey to the ordinary ear the idea of moisture or fluidity. The sound which comes nearest to it is the so-called *sub-crepitant* or *consonating râle*, which is much more characteristic of phthisis. It is a crackling sound, coarser and louder than the fine crepitant râle just mentioned, and is often only to be heard in deep inspiration or in coughing; it may be heard in both inspiration and expiration when well marked. Râles are also classified as small, medium, and large—a confusion of thought, since such features should be strictly confined to visual phenomena; but if such classification be accepted, the consonating râles may be said to be of small medium size. They are supposed to be produced in small bronchi which are enveloped by a layer of consolidated or infiltrated pulmonary tissue, and they are often audible over small cavities. As softening progresses and secretion becomes more abundant, these râles convey the sensation of 'bubbling,' and at a later stage, when excavation has advanced, the suggestion of a distinct 'gurgling' sensation is obvious.

The medium-sized râles are often very sparse, only one or two being audible during an inspiratory act, when they will then be heard at the end of inspiration or on coughing as one or two distinct 'clicks,' which, when taken with the dulness on percussion and the almost constantly present bronchial breathing, is pathognomonic of phthisis if heard over a moderately sized area near the apex of the lung.

When any of these râles are produced inside cavities, or in bronchi in the immediate vicinity of cavities, a distinct metallic character is added to them which is highly characteristic and which is usually associated also with amphoric breathing and increased vocal resonance. The coarser or so-called larger râles are very frequently heard both in inspiration and in expiration.

In contradistinction to the râles just described, which are usually defined as *moist* sounds of a rattling or crackling and non-musical character, produced in the air-cells, in smaller bronchioles, or in cavities, various kinds of *rhonchi* are heard in the phthysical chest. These have a more or less musical quality, and are usually classed as *dry* sounds—an unfortunate nomenclature, as pointed out by F. Taylor, since they are often caused by mucus lying in the tubes. Moreover, there may be said to be dry rhonchi and moist rhonchi, as there are dry râles and moist râles, all to be sometimes heard in the same chest in a long-standing case of phthisis, though many authorities confine the term 'rhonchus' to a sound which is always a dry one. The dry rhonchi are loud snoring or sonorous sounds of low pitch produced in the large bronchi, and may be present when a necrosing irritating focus of tubercle sets up bronchial inflammation after eating its way into a large tube. They are heard often in old fibroid cases and where there is much emphysema and spasm of the bronchi, as in asthmatic cases. The higher-pitched, hissing, whistling, or sibilant rhonchi are produced in the tubes of a smaller size, but not in the terminal bronchi, alveoli, or in cavities. The rhonchi are thus of little diagnostic signification, being evidence of coarse bronchial inflammation causing narrowing of the lumen of tubes of considerable size. They may be very transient when caused by plugs of expectoration lying in an otherwise normal tube, in which case they are dissipated by coughing. They are important, however, since by their loudness they may entirely mask the fine moist râles and the modifications of the vesicular breathing which are so characteristic of early phthisis. As they are usually heard over the entire chest in the catarrhal or bronchial condition, which is so apt at times to be associated with the early localised deposits of tubercle, a careful contrast of the auscultatory phenomena must be made at many different corresponding areas of the chest-wall, and the changes in them which follow upon coughing and forced respiration should be closely scrutinised.

Remonchamps, under the name of *Laryngeal crepitus*, claims to have discovered a method of auscultation which he believes is pathognomonic of pulmonary phthisis from its commencement. It is applied in the following manner by the physician, who stands in front of the erect patient; placing his right hand upon the patient's left shoulder and resting his left thumb on the patient's chin, he brings his left ear to the patient's mouth, keeping it at a distance of two or three inches, when a fine crepitating sound is perceived as if proceeding from the patient's larynx. The crepitation resembles the sound produced by a fine pen in slowly scratching

paper and is most marked in expiration, and it is heard throughout the entire course of the disease in all its stages.

In addition to the above modifications in the vesicular breathing and to the presence of adventitious sounds produced in the tubes, alveoli, and cavities, signs of pleural mischief are very common. A fine friction-rub at the apex is a very suggestive sound. A marked systolic murmur over the subclavian artery, heard below the clavicle, is sometimes caused by the increased power of conduction gained by the solidification of the apex. Cardio-respiratory murmurs and increased intensity of the heart-sounds may be due to a similar cause lower down in the lung.

The voice-changes, as heard through the stethoscope, are of considerable diagnostic importance.

Vocal Resonance is always increased, unless when there is a marked localised thickening of the pleura over the area of tubercular consolidation, or when a bronchus is more or less completely obstructed. When well marked, the increase is described as *bronchophony*, and it is usually heard over areas of consolidation which yield bronchial breathing; it is identical in all respects with the voice-sound heard over the bifurcation of the trachea, and sterno-clavicular joint. Whatever may be the acoustic explanation of bronchophony, whether it be a phenomenon produced by increased power of conduction or one due to some intensification of the sound-waves in the imprisoned bronchi, it may be taken as always meaning either consolidation of the lung or the presence of a cavity. Since in the latter case the voice-sound is always further modified and associated with other characteristics, the presence of bronchophony may be practically accepted as evidence of pulmonary consolidation; it is nearly always accompanied by increase in the vocal fremitus.

Pectoriloquy may be described as an exaggeration of bronchophony; it may be said to exist when the articulate sounds are distinctly heard through the stethoscope as if the separate words were spoken directly into the ear. *Whispering pectoriloquy* is the term employed when this condition is most typical, and it affords a more accurate method of demonstrating the presence of the conditions upon which the phenomenon of pectoriloquy depends. If the patient be made to whisper, the *whispered* words are distinctly heard through the stethoscope with abnormal clearness and intensity. Pectoriloquy and whispering pectoriloquy are indications of a considerable degree of consolidation surrounding a large bronchus near to the chest-wall or of a cavity in free communication with a bronchial tube. Pectoriloquy is also heard in phthisis when pneumothorax has developed and the pleural air-cavity is in direct communication with a bronchus; but it is worth remembering that it is occasionally present

over an airless lung which is compressed by extensive pleural effusion. The intensification of the voice-sounds is obviously always associated with a similar change in the sound produced by coughing—a condition known as *tussive resonance*. In pneumothorax and over large cavities the vocal resonance usually has superadded to it a distinct amphoric or metallic quality similar to that which accompanies the breath-sounds and the râles. Especially in pneumothorax, but sometimes in large partially filled cavities, it is accompanied by the phenomenon known as *metallic tinkling*, in which a few isolated metallic or amphoric râles are heard as if produced by the falling of a drop of liquid from the roof of the cavity into its liquid contents.

SIGNS OF A CAVITY.—These have already been detailed in the description of the examination by inspection, percussion, and auscultation, but they may be here advantageously recapitulated for the reader's convenience. In any description of the evidences of a cavity the signs detailed are liable to convey the impression that this condition is scarcely likely to be overlooked; nevertheless, the diagnosis of a considerable vomica in the lung is often beset with great difficulty, especially if it be situated at any considerable depth. The most frequent source of error lies in the proneness to attach undue importance to any one or more signs without a patient weighing of all the items of evidence afforded by the different methods of examination.

INSPECTION, as a rule, affords scant information; there may, however, be marked retraction of the area overlying the excavation and corresponding diminution in the respiratory movements. If any degree of cicatrisation has supervened in a large apical cavity the margins of the lung may be retracted from the cardiac surface, showing an extended area over which the impulse-beat is visible. In some cases the apex-beat may be displaced upwards and to the left, or it may be even slightly displaced to the right, though these alterations are more likely to follow in fibroid changes independent of vomicae. Very rarely is a transient fulness or bulging over the cavity observed during the act of coughing; but when this sign is well marked it is highly characteristic of superficial cavities.

PALPATION will reveal marked increase in vocal and tussive fremitus, unless this be diminished by pleural effusion; it will also confirm the evidence of retraction and diminished movement detected by inspection. Sometimes, owing to retraction of the lung, the closure of the pulmonary valves may be felt by the fingers placed over the second left space.

PERCUSSION over a cavity gives very variable results: if the cavity be deep or surrounded by a dense wall of airless infiltrated lung, the ordinary tap yields a dull sound, whilst

a heavier stroke may bring out a note of higher resonance than the normal. Usually a large cavity situated near to the surface, and without much infiltrated lung being situated between it and the chest-wall, will give a well-marked tympanitic note which frequently has a distinct amphoric or bell-like character when the walls of the cavity are smooth. The marked presence or total absence of this tympanitic note leads to confusion if other signs be not duly weighed, because there may sometimes be a cavity with normal pulmonary resonance over it, and a similar tympanitic note may be elicited when the pulmonary tissue and chest-wall are in a highly relaxed or yielding condition. In the normal state this note is elicited by percussing over the trachea and larynx. The following tests should be applied: The part should be percussed while the patient's mouth is kept open; and when a cavity is present the tympanitic note is markedly raised in pitch: this is known as Wintrich's test. Friedreich's manœuvre is to cause the patient to take a deep inspiration, when the pitch also rises so high that the note may disappear as the tension of the wall increases; upon expiration the pitch falls again. Gerhardt's plan is to alter the position of the patient during percussion, when the pitch will be observed to rise as the patient assumes the erect or sitting posture, and to fall again when he lies horizontally.

A well-marked cracked-pot sound is highly characteristic of a cavity, but must not be accepted as pathognomonic, since it may sometimes be demonstrated by percussing the normal chests of very young children when they are in the act of crying. If one coin be placed over a very large cavity and it be tapped with another coin while the stethoscope is applied to the back of the chest, a bell-like chime or echo may be heard as in pneumothorax (*bruit d'airain*).

A method of percussion has been recently introduced by Erni under the name of '*tapotage*,' from which he claims results of very remarkable value. Holding a flexible silver paper-knife by the end of the blade, he taps the chest by permitting the free end of the handle to strike the skin as the knife-blade is bent by a slight jerk of the operator's wrist, so that the gentle tap is followed by a rebound of the handle each time. As soon as a cavity is reached the tap immediately causes a cough, and expectoration follows in less than a minute.

AUSCULTATION reveals very variable but more reliable evidence of a cavity. If attention be first directed to a search for the normal sounds with the view of detecting modifications in them, the bronchial character of the breathing will be at once evident: it is low-pitched and hollow, and is frequently styled cavernous breathing. This may be easily mistaken for the high-pitched tubular breathing heard over

a pneumonic lobe, and Flint maintained that it could be differentiated from the latter by observing that the pitch of the expiratory sound was always lower than that of the inspiratory in cavernous breathing; whilst in tubular breathing the expiratory sound has a higher pitch than the inspiratory. The writer has tested this statement at the bedside by getting several observers with highly trained musical ears to discriminate the difference of pitch in well-marked cases of cavities, and he has been assured that the expiration was of higher pitch than the inspiratory in most cases. It is, however, usually true and comparatively easy of determination that the pitch of both inspiration and expiration in the tubular breathing heard over simple consolidation is much higher than that of cavernous breathing. It is possible that a firm thick wall or a surrounding zone of pneumonic tissue may raise the pitch of the expiratory sound in the hollow bronchial or cavernous breathing heard over a vomica. In the main, however, for all practical purposes tubular and cavernous and bronchial breathing may be safely regarded as an almost identical acoustic phenomenon described under different names.

When the cavity is large, the breath-sound, or bronchial breathing, has the amphoric or metallic character super-added; this is best marked when heard over an extensive vomica whose walls are smooth. It may be practically regarded as pathognomonic of a large cavity in the lung or of an open pneumothorax or pyopneumothorax.

The metamorphosing breathing already described is strong evidence of a cavity, especially if the expiratory sound ends in a soft, low-pitched, metallic murmur fading imperceptibly into silence after a harsh blowing inspiratory sound. This latter condition affords perhaps the most unmistakable proof of a cavity.

Bronchophony is present, and when this phenomenon presents the characteristics of pectoriloquy, and especially of whispering pectoriloquy, the increased vocal resonance being often accompanied by a metallic quality, the diagnosis of a cavity may be safely made.

The effect of coughing in bringing out the characteristics of the increased vocal resonance affords valuable information; the fluid in the cavity being churned up with the air, various bubbling, gurgling, and splashing sounds, or very coarse râles, are produced which exhibit ringing metallic or amphoric qualities. Even in the absence of these, the increased tussive resonance may become distinctly amphoric, especially over comparatively dry and smooth-walled cavities. Metallic tinkling like that heard in pneumothorax is highly characteristic, and it may sometimes be induced by coughing when it is absent in quiet breathing.

Post-tussive suction is the name given to the high-pitched inspiratory sound which is observed to follow coughing; it has been also styled 'the indiarubber ball sound,' and is obviously produced by the suction action of the elastic walls of the cavity after the forced expiration caused by coughing. In large cavities containing much fluid secretion and air, the physician's ear applied directly to the chest-wall as the patient's body is shaken may catch the splashing sound of Hippocratic succussion which is so characteristic of hydro-pneumothorax.

Cardio-respiratory or cardio-pulmonic murmurs are commonly heard over vomicæ, especially those situated in the left lung near to the heart, and they are always systolic in time; the heart-sounds may also be heard with great intensity. Sometimes the force of systole may set up splashing or other sounds in a cavity in close relationship with the pericardium.

Lastly, the different crepitant, sub-crepitant, or consonating, crackling, bubbling, and gurgling râles already described as more or less characteristic of the softening and cavernous stage of phthisis will be audible in most instances, either directly over the cavity or in the lung-tissue surrounding its walls. It is not the mere presence of these sounds which is indicative of a cavity, but the amphoric, metallic, and other qualities which the cavernous condition confers upon them.

It must be remembered that the physical signs of a cavity are always varying with the amount of liquid and air which it contains, and that at any time, even when the cavern may be comparatively empty of secretion, the leading bronchus may be obstructed temporarily by mucus, pus, or caseating foci, so that no breath-sounds are audible. A fit of coughing under such circumstances will usually change the entire picture—a fact especially to be remembered in cavities situated towards the base of the right lung, which when full of secretion may yield no physical sign save dulness on percussion, which cannot be differentiated from hepatic dulness. After coughing, or after the patient has been made to stoop so as to bring his thorax almost into the inverted position, the cavity becomes emptied and may yield a tympanitic resonance. Such cavities, however, are more frequently due to bronchiectasis; but they may be associated with phthisis, and are not infrequently met with in the very chronic fibroid form of the disease, which by its cicatrising tendencies causes considerable displacement of the heart and neighbouring organs, and pulls out the bronchial walls so as to produce great dilatations of the larger tubes.

In advanced phthisis, thoracometry, and spirometry usually give evidence of considerable departures from the normal standard, and the inspiratory pressure, as measured

by a mercurial manometer, shows some diminution early in the disease, but the expiratory pressure always falls later on.

Under Diagnosis the *serum-test*, *tuberculin-injections*, *opsonic index*, and the *diazo-reaction* will be referred to.

Much has been written about the different types or varieties of phthisis, and these may practically be regarded as caused by variations in the rate of the progress of the disease, the result of larger or smaller dosage of the specific bacillus, the varying degrees of natural resistance, or the supervention of complications. Clinically, phthisis has been classified as *acute*, *sub-acute*, or *chronic*; but this division of the examples of the disease is of little value, since the variety which may exhibit the most acute symptoms in the earlier stages of its progress often settles down into the most typically chronic variety, and a case which has commenced and progressed for a considerable period in an essentially chronic manner may suddenly assume all the features of the most acute form of the disease.

ACUTE OR FLORID PHTHISIS.—It is often possible to distinguish the type commonly known as *acute galloping consumption* or *florid phthisis* (which may remain distinct throughout its course) from the common chronic phthisis, the distinction, however, being mainly due to the rapidity of its progress, which sometimes ends in death in four or six weeks. This variety or type of phthisis is frequently described as the *acute lobular* or *bronchopneumonic* form. It is caused by numerous disseminated foci, which rapidly soften and break down, producing multiple small cavities with irregular walls scattered throughout both lungs, but more numerous in the upper lobes. It is identical in every respect anatomically and clinically with the acute variety of phthisis which supervenes upon the chronic when a large shower of bacilli invades the pulmonary tissue from the bursting of an old extensive tuberculous focus in some part of the apical region, causing a widespread infection by aspiration.

The onset may be abrupt, though probably in all cases a latent tuberculous focus has previously been in existence; and it is seen in its most typical form in patients who have recently recovered from an attack of influenza or have safely passed through a recent childbearing period. The symptoms and signs are, in the main, those of the ordinary form of phthisis, allowance being made for the wider or more disseminated distribution of the lesions, the fever, and other constitutional symptoms running a more severe and rapid course.

ACUTE LOBAR OR PNEUMONIC PHTHISIS is a rarer form which may also originate in a latent focus. Its onset is abrupt, with rigors, fever, pleuritic pain, dyspnoea, and cough with rusty expectoration, in every detail simulating an attack of

acute lobar pneumonia. The physical signs, too, are identical, and a diagnosis is not possible till after the expiration of the usual eight or ten days in which a croupous pneumonia runs its course. The fever, however, is much more irregular or irregularly remittent in the tubercular form, and the specific organism may be found in the expectoration. Sometimes an apparently typical case of acute lobar phthisis turns out to be an upper-lobe croupous pneumonia supervening in a patient whose apex had been previously the seat of tubercular deposit. The writer has witnessed a similar coincidence in the infection of an old tubercular lung with the specific pneumonia of influenza. Acute lobar pneumonic phthisis may terminate fatally inside twenty days; seldom does it last beyond a couple of months, unless in the very rare examples where the symptoms seem to burn themselves out inside two or three weeks, and the case then suddenly drops into the chronic category. Such instances may possibly be explained on the assumption of an ordinary pneumonia in a phthisical patient.

ACUTE MILIARY TUBERCULOSIS may affect the lungs without being a part of a general miliary tuberculosis; much more frequently it is associated with deposit of miliary granulations in the peritoneum, cerebral membranes, lymphatic glands, and elsewhere. In this latter case the constitutional symptoms, the brain-disturbances, and abdominal inflammation may overshadow or entirely mask the chest-symptoms, and death occurs before any physical signs develop in the lung.

Where the pulmonary tissue is alone or mainly invaded the disease has an abrupt onset, with high fever resembling the enteric type; the most prominent chest-symptoms are dyspnoea, the respirations rising to sixty or eighty per minute, with frequent hacking cough and considerable cyanosis. Expectoration is scant; later on it becomes rusty from pneumonic areas, or it may present the characteristics of ordinary bronchitic sputum, but rarely are tubercle bacilli found in it. When the bacilli are detected in the sputum they probably come from an old latent tuberculous focus in which the miliary invasion has originated. The pulse is very frequent, the emaciation extremely rapid, soon reaching an intense degree, and it is associated with great muscular prostration which is out of all proportion to the fever.

Miliary tubercles have been seen in the choroid, and occasionally the bacilli have been detected in the blood.

The physical signs may reveal nothing whatever wrong in the chest, or at the most some bronchitic rhonchi, scattered râles, and pleuritic friction-sounds, and occasionally an extensive tract of consolidation. As a rule, death takes place before the changes have proceeded so far as to give distinct evidence

of softening. In one interesting case watched carefully from day to day by the writer, the only symptoms observable were profuse sweatings, breathlessness, cyanosis, and cough; the physical examination revealed nothing but a generally distributed puerile type of breathing without a single adventitious sound; there was *no fever*, though a tuberculous focus was suspected in the abdomen. After death, which occurred from the exhausting toxæmia produced by the tubercular toxins, both lungs were found studded throughout their entire substance with fine miliary nodules so closely packed together as to leave apparently very little pulmonary tissue between them. Miliary tubercles were also found in different parts of the body and an old caseating focus in a cluster of mesenteric glands. Such cases are merely referred to here to complete the clinical picture of pulmonary tuberculosis; they are examples of acute miliary tuberculosis, and will be dealt with in the article on 'Tuberculosis.'

It must be remembered that chronic phthisis often runs, as might be expected, a more acute course in children. Being usually secondary to mesenteric tuberculosis, the physical signs are less frequently confined to the apical regions, and the disease shows a greater tendency to involve scattered areas in both lungs. In the aged the so-called senile variety is often observed; it is sometimes entirely latent and always runs a slow course, being generally associated with emphysema.

FIBROID PHTHISIS.—Of the varieties of chronic phthisis much discussion has always arisen about the acceptance, as a separate type, of the so-called fibroid phthisis. This is anatomically regarded as a fibrosis or cirrhosis of the lung, and, though often associated with tubercle, may certainly be found in its typical condition in syphilis, bronchiectasis, pneumokoniosis, and other states in which tuberculosis plays no part. It will be again referred to for convenience under 'Pneumonia,' the interstitial changes observed in the interlobular, interalveolar, peribronchial, perivascular, and subpleural fibrous tissue being still by many pathologists regarded as of a chronic inflammatory type.

When the cirrhosis is of tubercular origin the condition may be sometimes identified as supervening upon an ordinary chronic phthisis starting in the apical region. It may be practically regarded as an exaggeration of the fibroid changes observed in all cases of chronic phthisis in those areas in which the reparative process has been started. It tends, however, to slowly invade the lower lobe, and may ultimately involve both lungs. The pleura is enormously thickened, the lung-tissue is dense, tough, and airless owing to the great increase in its fibrous-tissue elements, and the bronchi are dilated.

the appearance of an unmistakable diazo-reaction, are most unfavourable signs.

Hæmoptysis, even when recurring often in small quantities, is not to be interpreted as necessarily of very unfavourable omen, though the profuse bleeding from an aneurismal dilatation on a medium-sized or large vessel must always be regarded as a serious symptom. The advent of moderate pleural effusion and the supervention of pulmonary emphysema in many cases appear to exercise an inhibitory influence over the progress of the malady. The involvement of the opposite lung to even a slight extent is obviously as grave as, or graver than, the evidence of the spread of the disease in the organ first affected.

The frequency of cough, amount of expectoration, and the number of bacilli in the sputum do not usually afford reliable data for prognosis, all these being sometimes negative, or present in slight degree, when the disease is spreading rapidly in the lung.

In the late stages, extreme breathlessness or dyspnœa, anasarca, albuminuria, profuse diarrhœa, evidence of amyloid disease, and laryngeal distress are often the heralds of a rapidly approaching fatal termination.

The question of the marriage of phthisical patients is a serious one often thrust upon the physician for his decision. It may be said that in the case of women the query should, as a general rule, be answered in the negative, the exception being in the case of women beyond the childbearing period in whom the disease has been quiescent for a considerable time and from whom a good family-history is obtainable. Whilst less restriction may be placed upon the marriage of males, the physician should certainly always protest against marriage as long as there is any evidence of activity in the disease, only giving his sanction when the chest and constitutional symptoms have been in complete quiescence for a period of at least a year.

Treatment.—*Prophylactic* or *preventive* measures are attracting more attention at the present time as regards tuberculosis than any other hygienic problem at any period in the world's history. This is the explanation of the steady and marked diminution in the still appalling death-rate from phthisis. Municipal authorities, by the dissemination of literature, have achieved much in the education of the public to the dangers of the conveyance of the disease by promiscuous spitting, by dried sputum, improper ventilation, &c.

By the regular inspection of lodging-houses and dairies, by the scrutiny of meat-supplies, by the enforcement of the State regulations regarding factories and workshops where unhealthy occupations are pursued, and by attention to the erection of more healthy houses and the destruction of ill-

lighted, imperfectly ventilated, and badly sewered tenements, much progress is visible everywhere. The importance of large open spaces in the midst of all densely populated districts is now universally appreciated. The adoption of notification has given public-health authorities much-needed information regarding the incidence of the disease in insanitary dwellings, as it also has given facilities for disinfection and other measures calculated to limit the spread of the disease. By the erection of sanatoria for the isolation of the poor in congested districts, and by the thorough cleansing of the streets of large cities after prolonged sprinkling with fresh or sea water, much, it is believed, has been done to diminish the risks of infection.

In the discussion of the etiology of the disease the writer has stated his views, based upon clinical experience, of the dangers of infection by the milk-supply, and though this is more or less generally recognised as a possible, or even probable, source of phthisis, nevertheless the more obvious and catching idea of the propagation of the bacillus through sputum has overshadowed in the professional and lay mind the due appreciation of the perils of an infected milk-supply. There is probably no combination of preventive measures which would more speedily and effectually curtail the ravages of phthisis than the establishment of a *strictly regulated municipal control of all milk supplied to city residents*. When it is considered that tuberculosis may exist in an advanced degree in apparently healthy and fat milch cows yielding abundant milk and living in the best-ventilated cowsheds and byres, the danger can hardly be exaggerated. The routine administration of the tuberculin-test can only be carried out under a scheme in which the municipal control is absolute. Now that it is accepted that bovine tuberculosis is capable of being conveyed to man, and the view that the bacilli only appear in milk when the udder is tuberculous is thoroughly discredited, there is good reason to hope that the milk-supply problem will soon be rationally grappled with. Great expectations surround the discovery by Behring that cattle can be rendered immune; and this, when taken in conjunction with the recent researches of Calmette and Guérin, already referred to, should prompt immediate action by the State.

Individual or private prophylaxis is indicated upon the same lines as suggest municipal interference. The disposal of the sputum is a simple matter; it should be directly passed from the patient's mouth into a spittoon containing water or a disinfectant, and it should be afterwards burned or rendered inert by filling the vessel with boiling water before emptying it into the house-drain, the main precaution being that the expectoration should never be allowed to dry

or come into contact with handkerchiefs, or be permitted to fall upon floors, dresses, or walls. Practically there is no danger to be feared from the exhalations of the patient's breath; but the risk of the dissemination of the bacilli in the spray thrown out in coughing is a real one, though minimised by many authorities. Free ventilation, abundance of sunlight, and rigid cleanliness will remove all danger of conveyance of the disease to healthy subjects when the sputum is carefully seen to; but the phthysical patient should have a bed and, when possible, a bedroom always to himself.

As regards individual prophylaxis in those suspected to be predisposed to the disease, there is no measure of such vital importance as the sterilisation of the milk-supply; and even in individuals already in the early stage of phthisis this should invariably be insisted upon. The writer has found that a very convenient method of carrying this out on a large scale in schools is to insert a pipe conveying steam to the bottom of each milk-can as it is brought fresh from the cowshed. In a few minutes the temperature of the milk can be raised to near the boiling-point by this plan; and although the milk becomes diluted by the condensed steam, all flavours such as those which adhere to ordinary cooking-utensils, &c., are avoided, and its taste is but slightly altered. The degree of dilution resulting from the employment of this method of sterilisation is equivalent to about one-fifth part of the original bulk of the milk. It is needless to say that a phthysical mother should not be permitted to suckle her infant.

The suggestion that all enlarged lymphatic glands should be excised is not worth consideration; the great majority of patients suffering from these never develop phthisis, and it seems certain that the disease is sometimes liable to supervene in an acute and serious form after such operations, especially when they are of but a partial nature, as is often the case in tubercular adenitis. Removal of the tonsils is frequently resorted to when these are hypertrophied and the seat of repeated attacks of sore-throat.

In the writer's opinion, few matters of detail approach in importance the necessity of maintaining the mouth in a healthy state by strict cleanliness and the removal of all necrosing teeth.

As regards the question of prophylaxis in an individual suffering from a small focus in the lung, the danger of his infecting the abdominal viscera by swallowing his sputum is always to be guarded against.

The selection of open-air occupations, change of climate, choice of food, the necessity of sleeping with open windows, and many other hygienic measures recognised in the routine

treatment of the disease, may be accepted as prophylactic agents applicable in all cases of delicate individuals believed to be predisposed to tuberculosis.

The *therapeutic measures* indicated in the treatment of phthisis are numerous, and the entire subject is a wide one. The author in his 'Dictionary of Treatment' gives a summary of the most important of these which covers twenty-five closely printed pages. Only the general principles of the therapeutics of the disease can be here referred to.

The first thing to be realised by the physician, and kept ever before his mind, is that pulmonary tuberculosis is liable in many instances to spontaneous cure. This fact of late years has been demonstrated in thousands of cases where the specific lesions of a former phthisis have been detected in the lungs of individuals dying from other causes, the tuberculous nature of the changes being recognised by the cicatricial tissue and calcification of caseated products in which all traces of the bacillus had ceased to exist. Again, cases are almost daily met with by physicians in extensive practice where no evidence of the disease can be detected, though the physical signs in the chest and the abundant presence of bacilli in the sputum formerly showed extensive foci of the disease. From abundant clinical data it is certain that in various well-recognised therapeutic agents the physician possesses the means of increasing this natural tendency towards recovery. If the tendency of the disease towards spontaneous resolution be regarded as identical with, or at least largely dependent upon, the amount of the natural resistance of the patient's tissues to the bacillus, the therapeutic indication will obviously be to increase this tissue-resistance. Other considerations are of secondary importance.

The main agencies upon which reliance is to be placed are an open-air life day and night, abundance of sunlight, the ideal proportions of rest and exercise, a well-regulated diet, and the utilisation of such agents as will ensure the maximum of nourishment being ingested and digested. As Cornet puts it: 'the present-day treatment of phthisis is essentially nothing more than a heightened hygiene of the body and mind.' No single element in the cure, according to this authority—neither air, nor food, nor hydro-therapy, nor medicine—affords any guarantee of success, this result being only obtainable by the satisfaction of all the physiological and hygienic demands of the human organism from the greatest to the least significant.

The *open-air* treatment, first insisted upon by Henry MacCormac of Belfast, and by Brehmer of Göbersdorf, is perhaps the most important of all the methods. The so-called *climatic* treatment of phthisis is but a phase of this, and, as long ago formulated by the writer, the rule may be

accepted that the climate which affords the greatest facilities for spending the largest amount of the patient's time in the open and, as far as possible aseptic, air is the one most likely to lead to the best results. To consider that climate possesses any very marked specific virtues over and above the advantages which accrue from the possibility of eating, living, resting, and exercising in fresh air and abundant sunshine, is to risk the patient's carrying with him into his new climate the habits of late hours, irregular feeding, and the various excesses which have probably reduced his vital resistance and rendered him susceptible to the inroads of the bacillus in the first instance. Thus the habit of attaching undue importance to the specific virtues of the atmospheres of high altitudes and ocean life often tempts the patient to omit the other necessary precautions, such as the proper regulation of the hours of rest and exercise, diet, medicine, &c. Phillip rightly observes that much of the beneficial influence of certain climates is hopelessly lost owing to the absence of intelligent appreciation or application of the major hygienic requirements.

The success of *sanatorium*-treatment depends upon the utilisation of the fullest possible opportunities of spending all the patient's time in the open air, whether during eating, exercising, resting, or sleeping, together with the most minute attention to all other physiological and hygienic demands; and abundant evidence is forthcoming from all regions which proves that good results are obtainable in almost every climate by attention to the above details.

Feeding comes next in importance to fresh air; increased nutrition retards the augmented metabolism which causes the emaciation of phthisical patients, and it affords at the same time a valuable means of increasing the natural resistance of the organism to the bacillus. The truth of this is seen in the frequency with which tuberculosis is observed to occur in subjects who have been under-fed or from any cause have been rendered unable to assimilate their food. If Osler overstates the importance of dietetic treatment when he says, 'make the patient grow fat and the local disease may be left to take care of itself,' the error is certainly on the right side. It is unnecessary to go into details as regards the constituents of the ideal dietary for the phthisical patient: fats and carbohydrates must be given in abundance; but individual peculiarities demand consideration, and the greatest variety should always be aimed at. Five or six meals should, if possible, be consumed during the twenty-four hours, and in some sanatoria this is insisted upon, though at Nordrach-Colonie but three meals are given—*i.e.* at 8 A.M., 1 P.M., and 7 P.M. Milk, cream, butter, and eggs should largely enter into the diet, and koumiss prepared by the writer's method is

a valuable adjunct. In dyspeptic subjects an almost raw-meat diet is often indicated at the start, when fats and carbohydrates or starchy foods are not tolerated.

One of the greatest advantages observed in the sanatorium-treatment is the remarkable improvement in the appetite which usually rapidly follows the new life in the open air and sunshine, the dyspeptic symptoms often vanishing in a few days after the patient has commenced to take his meals in the open air, and sometimes enormous quantities of food can be consumed and digested by invalids who had not indulged in a full meal for months previously.

Of the method of treating phthisis by *forced feeding* little need be said; Débove's *suralimentation* plan consists in introducing by a soft rubber tube large quantities of powdered meat, eggs, and milk into the stomach several times a day. It is seldom necessary, unless in neurotic cases where there is total loss of appetite co-existing with good digestive powers. The Weir-Mitchell plan is preferable, however, in such cases, and soon the neurotic patient becomes able to take his food without being forced in any way. Alcohol enters into most Continental dietaries in some form or other; but its administration must be closely supervised as the requirements indicate, and it is needless to remark that it should be sparingly, if at all, used in the case of children. As a rule, only as much should be prescribed as will aid appetite and digestion, and it should never be forced upon the patient.

Gymnastic exercises, massage, baths, hydropathy, mental occupations, sports, pastimes, and every other adjunct to treatment must be regulated with the view of improving appetite, and as a rule those which can be carried out in the open air should be permitted; thus bathing and swimming when climatic conditions are suitable, are preferable to ordinary hydropathic methods.

Rest being an essential condition in all cases where the temperature is elevated, the indulgence in any form of physical exercise is only to be permitted after careful thermometric observations have been made and studied and the course of the disease watched for a time. In all cases rest for at least half an hour before meals and for a longer period after each repast should be insisted upon. Breathing-exercises are often permissible before walking can be safely indulged in.

Clothing is an important item in treatment; the amount of apparel worn by phthisical subjects is as a rule injuriously great. Flannel should be worn next the skin or over a thin woven woollen garment of light texture. The hygiene of the skin must be carefully attended to, and this is practically an impossibility when the body is constantly enveloped in numerous layers of warm and heavy clothing. Obviously careful wrapping must be enjoined in the case of patients

resting in the open air when exercise is impossible on account of pyrexia or hæmoptysis.

Drugs should take a subordinate position in the therapy of phthisis, if we exclude cod-liver oil, which must rightly be regarded from the standpoint of a food. Tuberculin will be considered later on. In the main, the use of drugs is becoming more and more limited to the treatment of complications. Antiseptic agents of the volatile class administered as sprays and inhalations, with the view of being brought into contact with the diseased areas in the lung, are steadily passing into disrepute. They are clearly indicated when the larynx is involved.

Better reasons seem to be forthcoming for the administration of creosote and its derivatives—guaiacol, &c.; these, when taken by the mouth, appear to enter into chemical combination with the toxins of the bacilli and cocci, whose injurious effects they seem to minimise. The cinnamic-acid compounds either act in a similar manner or they stimulate the phagocytic mechanism; but no drug hitherto discovered seems to possess any direct bactericidal power when administered by the mouth or hypodermically in doses harmless to the human organism.

The above open-air, dietetic, and improved general hygienic treatment can be most thoroughly carried out only in well-managed sanatoria under the supervision of specially trained physicians; but it is a good plan to permit the treatment to be pursued for a few months in such an institution, after which the patient can be trusted in most cases to carry it out at home or under better climatic conditions, after his education in hygiene has been perfected.

After a few weeks or months, in early cases, the open-air and dietetic hygiene begin to tell upon all the symptoms and signs. As the appetite improves, the first proof of the patient's progress is seen in a marked increase of body-weight; sometimes as much as one pound *per diem* has been reported, and a steady gain of one pound per week for many months is a common event. Expectoration lessens and cough is markedly improved, the hacking unnecessary cough often entirely disappearing. The pulse-rate falls steadily, and it is soon observed that it does not rise markedly after mild exertion, as was formerly the case. Fever lessens and occasionally entirely disappears after some weeks, the night-sweats and chilly sensations vanishing as the temperature falls and the heat-centres become less sensitive. In a considerable percentage of cases the physical signs in the lung clear up, and in some they entirely disappear, leaving the patient quite cured. In some cases, however, the patient, though apparently well and even in an obese condition, is found to present little improvement in the physical signs of the disease;

such cases, though markedly relieved and able to pursue their former avocations without difficulty for indefinite periods, are prone to relapses. It is at present impossible to estimate the percentage of cases which can be regarded as permanent cures, statistics are so variable and contradictory; and none are as yet available upon a large scale where the classification enables an approximation of the value of the results to be made out. Chidell quotes Rumpf's figures as proving the value of classification: 75 per cent. of all his cases, where there was slight affection of not more than one lobe or of two half-lobes, were able to resume and continue work up to the period of reporting—*i.e.* for two or three years after treatment had been discontinued; whilst in the severe cases 54 per cent. were found to have died in the same period.

Climatic treatment is dealt with in the article already referred to. The view is gradually gaining ground that there is no specific curative element in any special climatic conditions. The rule above given may be safely accepted as a means of judging of the value of any given climate, always provided that the same rigid adherence to open-air life, improved hygiene, and dietetic aids be insisted upon. The stillness, rarity, and purity of the air in high mountain-climates are important aids in improving the natural resistance and increasing the breathing-power of the invalid; but the variations of temperature in the shade and sun often outweigh other advantages and test the endurance of the invalid too severely, especially when renal and laryngeal complications exist. Such high altitudes are more suitable in winter, owing to the lesser degree of temperature-variations and the greater stillness of the air. Mountain-climates at more moderate altitudes are usually best suited for summer residence, owing to the lesser variations of temperature at this season.

Lowland climates, when well wooded and sheltered by mountains, are most suitable in severe or acute cases, the changes of temperature being trivial and interfering but little with the working of the hyper-sensitive heat-centres. Patients who find the higher altitudes too stimulating often do well in such climates, though the appetite is usually not so good. Their value is enhanced by the absence of dust and often by the presence of balsamic emanations from the conifers in the adjoining woods.

Sea-coast climates are favourites with some patients who can eat and sleep best in the vicinity of the ocean, though others remain wakeful and suffer from anorexia. The sea-climate is obtainable in its best form in the ocean-voyage, in which the disadvantage of exposure to winds is counterbalanced by the absence of dust. It is very suitable for strong patients with limited or quiescent lesions, but only for

those who are good sailors and when due precautions are taken as far as possible in avoiding stormy latitudes and bad seasons. As in the selection of any shore or mountain climate, the essential value of the treatment must be measured by the number of hours daily to be spent on deck in the open air. In storms and rain at sea the effects on those who cannot sail without the dread of sickness are often disastrous.

The realisation of the fact that much more depends upon the hygiene than upon any particular quality of the air is markedly diminishing the numbers of patients who are sent long distances from home in the advanced stage of the disease.

Cornet sums up with the following conclusions about climates on the treatment of phthisis: 'Cold dry rarefied air stimulates strong individuals, increases the appetite, and also at times the cough; upon weakly feverish patients it often produces insomnia and diminishes appetite.' Such cases should first try the less stimulating low-lying or sub-alpine regions during the summer before venturing upon the higher and more trying rarefied-air climates in the winter. Hæmoptysis and albuminuria, especially when co-existing with hectic fever, are more suitable for the lower-level climates always. Moist warm climates are most suited to phthisical patients who suffer from bronchial catarrhs and laryngeal trouble.

Tuberculin-treatment.—The injection of large doses of Koch's original tuberculin was followed usually by violent reactionary fever and often by acceleration of the progress of the disease, and not rarely by a speedily fatal issue. These results led to its almost entire abandonment, save for diagnostic purposes, till the brilliant researches of Sir A. Wright placed the tuberculin-treatment of phthisis upon a rational basis.

The explanation of the value of what is called the *opsonic index* has been described upon page 1342, under Diagnosis.

Wright found that the depression of the opsonic index which followed large doses of Koch's original tuberculin was fraught with great danger. In the case of *systemic* infection a material increase of the toxins already freely circulating in the blood, such as would result from the old doses of tuberculin, invariably does harm; hence the newer method of using the drug by even minute doses should at present be confined to strictly *localised* tuberculous infections. This at once puts all cases of pulmonary tuberculosis with a marked pyrexial character outside the category of safe treatment; but Wright believes that such examples of the disease may be safely and oftentimes successfully treated by tuberculin when long periods of rest, suitable diet, and other hygienic measures have terminated the series of auto-infections which follow exercise; the cases are then practically to be regarded as instances of localised infection.

In the main, therefore, this form of treatment, whose value is established beyond doubt in such localised affections as lupus, tubercular bladder, and joint-disease, lymphatic-gland infections and peritonitis, is practically confined to the cases of phthisis of an *apyrexial type*.

The essential aim in the treatment is to raise the opsonic index, which, in other words, is equivalent to increasing the natural resistance; and Wright maintains 'that an increase of the opsonic power of the blood can be achieved and maintained by the inoculation of a series of appropriately adjusted and interspaced doses of tubercular vaccine.'

Koch's new tuberculin R. is always employed, the initial dose never exceeding the equivalent of $\frac{1}{8000}$ mgm. of the tubercle powder, which is raised afterwards to, but never above, $\frac{1}{3000}$ mgm. Doses beyond these always give inferior results, and hence the marked discrepancies in the opinions of various physicians who use the remedy in smaller, but more frequently in larger, amounts. The *negative* phase after these small doses becomes less and less marked with each injection, and there is never any constitutional disturbance; a new dose should not be administered till the effects of the previous one are passing off, as tested by the opsonic index. Once a fortnight is often sufficient, as at this interval the positive phase is on the wane, and a slightly higher level than the normal may be almost continuously maintained; but the proper intervals between the doses can only be determined by results. The duration of the treatment can only be decided upon after numerous observations of the opsonic index. Ross considers that patients possessing an opsonic index above 1.0 are not suitable for the treatment, as in these it may be safely inferred that nature is doing her utmost. The above fractions are Wright's 'corrected' doses.

The theory of Wright explains the difference between the results obtainable in localised and systemic infections. He regards the bacilli in the former case as developing in the tissues involved in a region of *lowered bacteriotropic pressure*, the anti-bacterial substances being present in less amount than in the blood, and the injections afford a means of increasing the amount of these substances in the lymph-stream.

Klebs employs a preparation called antiphthisin, in which the bactericidal substances in the tuberculin are alone retained, the other objectionable and fever-producing toxins being separated by chemical processes. His method is certainly harmless, but it remains to be proven that it is as efficacious as the tuberculin-method.

Serum-therapy.—This method of treating tuberculosis is associated with the name of Maragliano, who maintains that it is possible to immunise animals against the bacillus of tubercle and from their blood to prepare an antitoxin for

administration to the human subject of phthisis. The immunising injections given to the animals consisted of various toxic substances prepared from pure cultures of the tubercle bacillus. It appears certain that with the immunising methods at present at our disposal serum-therapy is practically a failure ; but there is room for hope that the more thorough methods of Behring will ultimately lead to the production of a serum which will constitute a specific treatment of phthisis.

The treatment of the symptoms and complications of phthisis affords a wide field for the therapist ; these, when not controlled by the open-air and dietetic plans, must be met by remedial agents employed upon the usually recognised principles. The remedies indicated for the relief of high fever, cough, sweating, pain, diarrhœa, hæmoptysis, &c., are discussed in detail, along with a brief reference to the drugs hitherto employed as routine agents in the treatment of phthisis, in the author's ' Dictionary of Treatment ' (4th ed.), pp. 689-713. Treatment by congestive hyperæmia has been recently adapted to the lung by the use of Kuhn's mask.

PICA

This is the name given to the disordered or perverted state of appetite met with in dirt-eaters. The condition is one nearly always associated with some psychosis. It is very common in asylum-patients, the most frequent phase of it being clay and cinder eating. Similar cases are met with amongst hysterical patients who keep near the borderland of insanity. Imbeciles and idiot children betray often the propensity of putting into the mouth anything coming within their reach ; this is different from the perversion of appetite which is seen in highly neurotic children and epileptics who manifest a desire to eat special articles, like paper, blacking, soap, grass, sawdust, cotton-wool, &c. Closely allied to this abnormal or degraded form of appetite is that met with sometimes in pregnancy. In the pregnant condition true dirt-eating is not very rare, but much more frequently the perversion rather takes the form of marked capriciousness of appetite, as seen in the yearning to eat raw meal, rice, tea-leaves, potato-parings, &c.

In children pica is often regarded as being due to round-worms in the intestines ; possibly in all cases the presence of the parasites is the direct result of the dirt-eating and not the cause of it.

The treatment of the condition must be carried out upon moral lines ; but obviously all catarrhal states of both bowel and stomach should be attended to.

PINTA

This is a contagious skin-disease, common in the tropical parts of North and South America, produced by the presence of moulds or hyphomycetes ; it is known by a variety of names in the different localities in which it prevails, as Pannus Carateus, Spotted Sickness, &c.

Etiology.—The disease affects the native races more frequently than Europeans. It is spread by contact and flourishes amongst the filthy of all ages, those having any eczema or dermatitis being especially liable to it. It does not flourish at high elevations, but requires a hot atmosphere laden with moisture. Several distinct moulds have been detected in the scrapings from the scaly patches, as penicillium, trichophyton, pictor, monilia, aspergillus, and Gastambide's fungus. These may be found by examining the fresh scales after immersion in caustic-soda solution, the oval conidia being easily visible, with their branched mycelial network in the epidermal cells.

Symptoms.—The disease affects all parts of the body except the soles and palms, appearing first as scaly patches of white, red, blue, or black colour, which extend slowly and may involve a large extent of the entire skin. In the same individual patches of all colours may sometimes be observed, or one colour may predominate throughout the entire course of the disease, which is always most chronic, or permanent if not thoroughly treated.

In the white variety of pinta the patches resemble leucoderma, but are not surrounded by the deeply pigmented border, and have not convex margins. There is little itching or desquamation in this form, though the hairs bleach and are shed.

The red variety resembles pityriasis versicolor, but it is differentiated from that disease by its deeper site in the skin, not being removable by scraping or scratching ; when upon the face it may simulate lupus erythematosus. It only attacks white-skinned people, and causes considerable itching and sometimes ulcerations, whose discharges may be very offensive.

The black and blue varieties simulate tattoo-marks, but they spread rapidly and may involve a large area of the body.

Treatment.—Iodine, chrysarobin, and nitrate of mercury ointments are efficacious ; but once the moulds have reached the Malpighian layer treatment is difficult and sometimes futile.

PIROPLASMA HOMINIS

This is the name given to the parasite which causes a disease commonly known as Tick Fever, Spotted Fever, and Blue Disease, which is met with in the Rocky Mountain districts of the United States in the spring and early summer months. The parasite is allied to the *Babesia*, an unpigmented hæmoprotozoon which causes Texas cattle-fever.

Etiology.—Persons of all ages are attacked, especially those whose occupations expose them to the bites of the various ticks, which are most active in the spring seasons in the regions where these pests abound.

The hæmatozoon possesses slow amœboid movement, and is allied to *Piroplasma canis*, *P. ovis*, and *P. bigeminum*, whose life-history has recently been studied by Chowning. The early stage of the parasite in its coccoid and diplococcoid forms is seen in the human plasma and red cells. In its later stage it occupies a considerable proportion of the red disc, like the malarial parasite.

The theory that the hæmatozoon gains its entrance to the human blood by the tick is rendered probable by the investigations of Chowning and Wilson, who obtained a history of recent tick-bites in all the cases which they examined, though this is denied by Stiles. Koch has described forms of a parasite which he has traced from the stomach to the eggs and larvæ of ticks.

Symptoms.—In the Montana Valley the disease is seen in its most serious form, commencing with an abrupt invasion ushered in with chills and high fever. A typhus-like eruption, commencing in discrete rose-coloured macules, appears on the wrists and ankles upon the second or third day. In severe cases, according to the description of Chowning, these become petechial and confluent, covering the entire body; the temperature steadily rises, and the constitutional symptoms assume the typhus type. By the seventh or eighth day, just before the crisis, with a temperature of 107°, a pulse of 150, and a respiration-rate of 60, the patient becomes comatose and may sink, or a slow lysis inaugurates a tedious convalescence. Albuminuria is sometimes present, and local gangrenous patches upon the elbows, knees, scrotum, ears, or finger-tips are common in the early stages. The blood shows diminution of the red discs and slight leucocytosis.

In cases which end in recovery desquamation sets in about the fifteenth day, after the eruption has faded.

The death-rate is about 75 per cent., the majority of the cases succumbing between the sixth and eleventh day.

In Nevada and Wyoming a milder type of the disease is seen, in which there is no eruption and recovery almost always

Treatment.—No agent has been discovered which produces any effect upon the disease. The authority from whose description the symptoms have been above summarised recommends quinine in large doses.

PIROPLASMOSIS (KALA-AZAR)

The fever is severe, long-continued, and irregularly remittent, sometimes with complete remissions. It is always associated with a very considerable degree of anæmia and hæmorrhages from the mucous membranes, and purpuric spots on the skin. The clinical picture in the advanced stages of the disease, which is very often fatal, resembles the malarial cachexia, with its emaciation, pallor, firm large spleen, and often enlarged liver, and hence it has been sometimes described as Tropical Cachexial Fever. The progress is one of many months, the great majority of the victims being cut off by dysentery or sinking from marasmus.

Much interest has attached itself to the pathology of Kala-azar since the discovery of the organism by Leishman and Donovan, which is now known as the 'Leishman' or 'Leishman-Donovan body.' The parasite, according to Rogers, belongs to the genus *Herpetomonas* L  ger, the intra-corporeal and extra-corporeal forms of which have been identified, and are considered by Manson as possibly representing respectively asexual and sexual forms. The intra-corporeal form, or Leishman body, is a small oval organism, measuring, according to Manson, 2 to 4 μ in diameter, which, when stained by Leishman's method, shows two lilac-coloured chromatin-masses enclosed in a cytoplasm. The parasites are always intracellular, appearing in the endothelial cells of the blood-vessels and lymphatics in enormous numbers, in the spleen, liver, kidney, lung, bone-marrow, skin-lesions, &c. The parasites have not been transmitted to animals, though easily cultivated outside the body, where they never develop an undulating membrane like the trypanosomes. The failure is

probably owing to the fact that the parasite must be conveyed by some species of tick, bug, or other insect, as demonstrated by Patton, who witnessed flagellated forms in a bug which had sucked the blood of a Kala-azar patient.

The blood of patients affected with Kala-azar, in addition to exhibiting a few sparse Leishman bodies in the white cells, shows marked anæmia, the red discs falling sometimes to 2,500,000 per c.mm. The remarkable phenomenon of leucopenia is also observable, as in some cases of typhoid fever, the number of white corpuscles falling as low as 1 to 4,000 red cells, instead of the normal proportion of 1 white to 500 or 600 red. The main falling off is in the polynuclear cells; the large mononuclear leucocytes and the lymphocytes are also diminished in number, but are relatively more numerous.

Manson emphasises the importance of not accepting the synonym of tropical splenomegaly for Kala-azar, since some of the cases exhibiting large spleen, fever, and the other symptoms never exhibit Leishman bodies. In every doubtful case he insists upon the importance of scrutinising every leucocyte in the stained blood-film, with the view of detecting the parasite before resorting to splenic puncture.

PITYRIASIS

This is the name given to a variety of totally different skin-affections in which the most obvious symptom or sign is branny desquamation.

Thus *Pityriasis Simplex*, which most frequently manifests itself as a pityriasis capitis, is now universally regarded as a dry seborrhœa, whether appearing upon the scalp, face, or body, being produced by a local irritation of the sebaceous glands, resulting in an increased and altered secretion of their more solid fatty constituents.

Pityriasis Rubra should be regarded as a synonym for dermatitis exfoliativa, and is described under its own heading.

Pityriasis Rubra Pilaris, also known as lichen pilaris, is referred to under 'Keratosis.' It is allied to ichthyosis, being produced by an accumulation of horny cells, which form small papules round the orifice of the hair-follicles. Kaposi regards it as a form of lichen rubra acuminatus.

Pityriasis Rosea is an inflammatory condition characterised by a pink scaly rash, supposed by some dermatologists to be produced by a parasitic organism, though the disease is not contagious. It begins, according to Brocq, in an initial patch on the trunk, neck, or arm. The patch is oval or circular, with bright-red slightly raised edges and a dull-brownish somewhat depressed centre, which at first might

be mistaken for ringworm, as the patch extends at its margins and fades in the centre. After six or seven days, when the surface is seen to be covered by fine scales, other red or pink spots appear, generally upon the abdomen and neck; these may remain as maculæ, but more frequently they develop into round patches, resembling the primitive one, which extend at their borders, forming a ringed eruption covered with fine branny scales and associated with a variable degree of itching. The outlines of the patches, as these extend, join with those of neighbouring rings, giving a circinate or gyrate appearance to the rash (*P. circinata*), which in three or four weeks may cover a considerable extent of the trunk, upper arms, neck, and thighs. After a few weeks' further duration the branny desquamation diminishes, the wavy lines of the extending borders fade, and resolution is complete. The disease may appear at any age, but it is most frequently seen in children and young adults.

Diagnosis.—*Psoriasis* is excluded by the fineness of the scales, which never assume the silvery appearance of the plaques seen in that disease. Moreover, the site is different, the front of the knees and elbows generally escaping, and when the scales are scraped gently off the base does not reveal the red, vascular, or hyperæmic papillæ found under the psoriasis-patch, and the eruption in its comparatively short duration covers a much more extensive surface of the body than that which is involved in a recent psoriasis. The detection of the initial patch and of the maculæ scattered amongst the gyrate or circinate areas and the natural tendency towards resolution are also characteristic features in the disease which should prevent errors in diagnosis.

From *tinea* the disease is easily distinguished by its wide distribution and by the absence of the specific fungus. From *P. versicolor* its short duration and the absence of the easily recognised microsporon furfur differentiate it.

Syphilitic eruptions are excluded by the absence of a history of infection and of the specific lesions of the secondary stage.

Seborrhæa corporis is eliminated by the fact that it is an essentially chronic disease which never affects the limbs, but remains confined to the centre of the chest and middle of the back between the shoulder-blades; it always commences by the appearance of small red papules, which coalesce into patches, and it is seldom seen except in those who perspire freely and who wear heavy flannel garments.

Treatment.—Owing to the short-lived nature of the disease and its invariable tendency towards spontaneous resolution, the only indication is the use of some bland unirritating ointment or weak alkaline lotion to allay itching.

Pityriasis Versicolor, more frequently styled *Tinea Versicolor*, is a cutaneous affection caused by the growth of a

fungus—microsporon furfur—in the superficial layer of the skin of the trunk and adjoining portions of the limbs. Very rarely does it affect the face. It may appear upon patients at any age, but it is seldom seen on children; it is very common upon male phthisical patients and weak individuals who are overclad with flannel and perspire freely. The patches are of a yellowish-fawn or tan hue; the staining is so superficial that it is easily removed by scraping off the uppermost layers of the epidermis with the finger-nails or a stout brush. The scrapings consist of epidermal cells with botryoidal masses of strongly refractile spores, surrounded by a lattice-work of mycelial threads. The individual patches grow slowly, and with their slightly raised scalloped or convex margins may cover a considerable area of the entire chest, whilst the surrounding healthy skin may exhibit small circular islets of the same scaly and yellowish-brown character. In dark-skinned individuals the area affected may be so extensive and the staining so deep as to simulate bronzing. There is very little itching or discomfort of any kind associated with the growth of the fungus. It may be conveyed from one person to another, but only by close and prolonged contact, probably also by the wearing of a soiled flannel vest.

The treatment is simple, frequent cleansing by soap and water being usually sufficient; but an immediate removal can be accomplished by any antiseptic or bleaching agent, as a weak solution of chlorinated soda, sulphurous acid, or hypsulphite of soda. One or two paintings with tincture of iodine will also remove it.

Similar patches, but of darker colour, are sometimes observed in the regions where intertrigo is common; they are produced by microsporon minutissimum, and usually show more marked scaliness and considerable itching. This condition is termed *Erythrasma*, and is easily removed by iodine applications.

PLAGUE

This is an acute infectious malady characterised by the swelling and matting together of the lymphatic glands in different parts of the body, the result of a specific inflammatory process caused by the plague-bacillus, the presence of the buboes being often associated with septic, hæmorrhagic, and pulmonary changes.

Grave epidemics of plague under various names have been recorded in history dating back nearly to the advent of the Christian era; most of the pestilence-epidemics of the first centuries being almost certainly caused by this disease. The Justinian plague, commencing in the sixth century and

spreading over the great Roman Empire, lasted for more than half a century. The Black Death pestilence of the fourteenth century removed a quarter of the human inhabitants of Europe, and the Great Plague of London caused frightful mortality about the middle of the seventeenth century. Numerous epidemics in different parts of the world have been recorded since that time down to the Thibet epidemic in 1894, which, reaching Canton, Hong-Kong, and Bombay, swept over extensive areas in India and China and caused great destruction to life in 1902, more than two millions of deaths being recorded in India between this latter date and 1904, the healthiest as well as the most insanitary regions being from season to season overrun by it. In these late years the disease has appeared at Oporto and Northern Egypt, the Philippines, South Africa, South America, Australia, and small localised outbreaks have been witnessed at Glasgow, Liverpool, and San Francisco.

Etiology.—The specific microbe of plague was discovered in 1894 by Yersin. Known as the *bacillus pestis*, it is a non-motile, short, oval bacillus with blunt ends, often occurring in fresh culture in pairs, occasionally in chains, and, in older specimens, singly. It stains readily by methylene-blue and is decolorised by Gram's method. Its growth in various media is highly characteristic; it does not produce spores, and, although aerobic, it can be cultivated under anaerobic conditions. Though subject to variations, its average length being slightly under $2\ \mu$, it is easily and positively recognisable, according to Hunter, by its bipolar staining. The bacillus is found readily in the enlarged glands and in the œdematous fluid surrounding them, in the blood, bile, sputum, lungs, and other internal organs. It has been frequently detected in the dust of houses where plague-victims have resided, and its presence has been demonstrated by many observers in the blood of domestic animals, as cats, dogs, and monkeys. Flies, lice, bugs, and fleas have been found to harbour the bacillus, and it appears to be at least highly probable that the disease may be transmitted to man by these parasites.

Rats, both the grey and black varieties, but especially the latter, are highly susceptible to the disease, and undoubtedly play an important rôle in the spread of plague, as will be referred to presently. Mice are also believed to act as carriers of the contagion.

The bacillus can obviously find its way into the human body in food infected by the excreta of the rat and by insect-contamination, but it is much doubted whether the disease can be spread in this manner. No direct evidence of infection by the gastro-intestinal route is forthcoming, though Hunter and White maintain that the disease is usually transmitted

through the gastro-intestinal surface. Some authorities regard the tonsil as the probable portal in the cases where the cervical glands are chiefly involved. Inhalation of the bacilli is an undoubted source of the disease in the pneumonic cases, and the presence of the organism in the sputum affords an easy explanation of its transmission to healthy individuals. This is strengthened by the fact that the pneumonia of plague is highly infectious, the disease occurring in the pulmonary form in those brought into contact with the pneumonic cases in hospitals.

In the majority of cases the bacillus gains its introduction to the human body by *inoculation*, the point of entrance being some trivial wound, abrasion, or insect-bite. The frequency with which the lymphatic glands of the groins are affected was considered by the Indian Plague Commission to be explained by the very great area of the skin drained into the inguinal vessels. The explanation of the inoculation being produced by the bites of fleas which have escaped from the fur of rats suffering from plague is insisted upon by Simond. There is some difficulty in accepting it as a working hypothesis for the rapid spread of plague, owing to the fact that the rat-flea does not readily bite man. Low states that the constant experience of the sanitary officials who disinfected the Cape Town houses in which plague had occurred was to be bitten by fleas, though few became smitten with the disease. He points out, however, that most of the officials were protected by Haffkine's prophylactic. The result of his experience he summarises in the conviction that the common method in which the rat infects man is by contact of the human abraded skin-surface with material infected by plague-rats.

There cannot be any doubt about the important part played by the rat in the dissemination of the disease, the investigation of many outbreaks revealing the fact that before the epidemic attacked mankind in the several districts the rats had suffered from the disease. Once, however, the malady has become epidemic, it would appear that the rat plays a minor *rôle* in its spread. The bacilli in the sputum, in the discharging secretions from the buboes, in the fæces and urine of plague-victims suffering in ill-ventilated filthy houses, where the bed-clothes and wearing garments become soon soiled and infected with the micro-organisms, afford obvious facilities for bringing the skin of healthy individuals into close contact with the origin of the disease. Under such circumstances, in the absence of personal cleanliness, it is obvious that the bites of flies, fleas, and other insects, with or without the abrasions caused by the finger-nails in scratching, afford ready methods for the introduction of the bacilli to the blood.

Filth, foul ventilation, and overcrowding are powerful etiological factors, as demonstrated by the immediate check which an outbreak receives when these causative agencies are removed.

The disease attacks persons of every age, and as a rule the epidemics are most severe and frequent during the hot season.

In the spread of the disease from place to place rats can usually be demonstrated to play a very important part. This holds true especially for the long sea-routes; examples of the spread of plague by the importation in ships of infected grain and merchandise of various kinds are now regarded as instances of the spread of the disease by means of the plague-rats which accompanied the cargo. Most frequently the disease in these cases makes its appearance in the rats living at the docks and landing-stages, and shortly afterwards the dock-labourers become affected.

Symptoms.—Several types of the disease exist, and though a particular epidemic may be distinguished sometimes by the preponderance of a particular type, it is usual to find all the forms of the disease represented, and, moreover, it is not uncommon to find the symptoms and signs supposed to be characteristic of each type all present in the body of a single victim to the malady.

BUBONIC PLAGUE is by far the most frequent form. There is a severe and acute invasion, setting in after an incubation-period of three to five days. One or more rigors or chills are followed by high fever, backache, headache, and pains in the groins or armpits; there is marked prostration, restlessness, and mental depression, and a feeling of being seriously ill. Hunter lays stress upon the severity of the headache, which is accompanied by giddiness, vomiting, and incoördination of speech, which resembles alcoholism. In some cases prodromal symptoms anticipatory of the above are observed for a day or two before the onset of the fever. Upon examination even a few hours after the invasion some tenderness may be elicited by pressure over the glands in the groin, neck, or axilla. In most cases the characteristic swellings may be felt before the expiration of forty-eight hours. The fever reaches its maximum by the third or fourth day, when it may register 106° ; it usually drops a few degrees, and remains stationary till the fifth or sixth day, when in fatal cases a record of 110° may be reached. The pulse is very rapid and dicrotic, the tongue dry and brown after being moist and coated, the respirations are accelerated, and thirst is marked. Mental symptoms become prominent, delirium resembling acute mania being common. There are often symptoms of muscular incoördination, which, as already stated, may affect the speech; the eyes are deeply congested, iritis and keratitis sometimes following.

Hæmorrhages into the skin, showing as small petechiæ or large dark purpuric spots, are often associated with extensive bleeding from any or all of the mucous surfaces.

The changes in the lymphatic glands proceed rapidly. There is marked pain and swelling; several glands may be found enlarged, matted together, and surrounded by a zone of œdema and by some hyperæmia of the skin, the whole forming a tumour or bubo larger than a turkey's egg before the expiration of twenty-four hours after the seizure. The lymphatics of the groin are far more frequently involved than those of other regions. Some obvious bubonic swelling is present in one or more glands somewhere throughout the body in more than three-fourths of all cases of plague. In the bubonic type now under consideration there is always a bubo to be detected when carefully searched for, and in the great majority of cases several are present, scattered throughout the lymphatic areas.

The death of the patient occurs before resolution or suppuration has had time to set in in most of the acute severe examples of the disease. When life is prolonged beyond ten or fourteen days suppuration may set in, the entire mass sloughing out or becoming gangrenous, or the bubonic swellings in one region of the body may break down and discharge, whilst others resolve and disappear. Sometimes, as observed by Low in the Cape Town epidemic, a bubo has been known to suppurate after the patient had been discharged from hospital as cured.

If the area drained by the lymphatics be examined carefully, sometimes a small papule or pustule may be found marking the site of the original inoculation, and such pustules contain the bacilli in numbers. From the papule or pustule inflamed lymphatic vessels may be sometimes traced by their red lines up to the enlarged gland; but as a rule lymphangitis is absent except in the lymph-vessels leading from the small intestine—a fact used by Hunter to prove that the bacilli gain admission through the intestinal tract. Other pustules of a similar character may spring up in the skin anywhere, but more frequently in the immediate vicinity of the buboes. These secondary pustules commence as papules and may finally take on the characters of carbuncles, exhibiting a firm dark eschar through one or more openings in the skin.

When resolution occurs, the buboes gradually shrivel up, and the patient recovers within two months; convalescence is protracted in the suppurative cases, sometimes for several months, owing to the slowness with which the ulcers or incised glands cicatrise.

Death may sometimes be witnessed as early as the first day, almost before time permits of the formation of buboes. Epidemics of plague resemble those of cerebro-spinal

meningitis in the frequency with which victims may be smitten down and die within a few hours without showing any specific symptom or sign of the disease save those caused by a profound toxæmia of indefinite type. The largest number of deaths in bubonic plague occur about the fourth day after the first symptoms of the invasion; generally the immediate result is due to heart-failure. Those who survive the first week may succumb from the supervention of the pneumonic or septicæmic upon the bubonic type of the disease.

PNEUMONIC PLAGUE.—It is not an uncommon event in bubonic cases to witness the supervention of pneumonic symptoms and vague pulmonary signs, with a rusty sputum which swarms with the plague-bacillus. The *primary type* of pneumonic plague is comparatively rare, being often entirely absent in many epidemics, whilst in some it occurs with frequency. The advent is similar to that of the bubonic variety, only the onset is more abrupt, the fever higher, and the general prostration more marked. Pulmonary symptoms set in with embarrassment of the breathing, pain in one side or in both sides of the thorax, with cough and expectoration; this latter is at first tenacious and scanty, but soon becomes copious and blood-stained, or consists apparently sometimes of pure blood, and is always laden with the bacilli as if in a pure culture. The thoracic signs are usually lobular and they may be comparatively vague; always they are much less significant than the grave constitutional symptoms, which usually run rapidly into delirium and coma, cutting short the patient's life in three or four days. Where he survives for a few days longer bubonic swellings may be detected in the groins, neck, or axilla. Few patients recover.

SEPTICÆMIC PLAGUE.—As just mentioned, secondary pneumonic symptoms often supervene in bubonic plague; so also the secondary fever is liable to eventuate in septicæmia or pyæmia. The term 'septicæmic plague' is, however, reserved for those primary and rapidly fatal cases of the disease in which there is profound toxæmia with delirium and coma, accompanied by a remarkable degree of collapse and cardiac failure, without the appearance of any bubonic swellings in the groin, neck, or axilla, though, as in the other types, enlargement of the liver and spleen may be detected. In this form the diagnosis is made from the abrupt and severe invasion, high fever, and rapidly fatal course of the disease; the plague-bacillus can be got in a sample of the blood removed from the finger-tip, and, moreover, severe hæmorrhages appear sometimes before the expiration of the first or second day.

Hunter only recognises two types of plague: (1) Primary plague-pneumonia, and (2) Plague septicæmia, with bubonic manifestations occurring as complications.

MINOR PLAGUE OR PESTIS MINOR.—These are the names given to a condition in which the main signs consist of some enlargement of the lymphatic glands, chiefly of the inguinal region, and the only symptom is a trifling malaise, usually associated with a very mild degree of fever. Occasionally the buboes suppurate. Pestis minor seems to be clinically identical with so-called *Pestis ambulans*; it has been seen several times noticed as a mild epidemic which preceded the outbreak of a pandemic of plague. There is little doubt but the buboes are caused by an attenuated form of the specific bacillus, and as a plague-epidemic is passing away similar buboes are not uncommonly met with in individuals who are unaware of any feeling of illness. The writer once saw the typical condition in a medical man who had inspected a ship in which a case of plague had occurred some weeks previously, though no persons at the port were afterwards known to suffer in any way.

During the height of an epidemic similar mild indolent buboes may appear in individuals who are able to continue their ordinary avocations, even after suppuration of one or more glands has taken place; these ambulatory cases are undoubtedly examples of true bubonic plague occurring in individuals who have either received an infinitesimal dose of the bacilli or in whom the natural resistance of the tissues is exceptionally well marked.

Other types of plague have been described as the *hæmorrhagic* and *intestinal*. In the former the cutaneous petechiæ and bleedings from the mucous membranes have been the obvious features, as in the Black Death epidemic of the fourteenth century; and in the latter diarrhœa and enteric symptoms have overshadowed the ordinary bubonic symptoms.

Morbid Anatomy.—The changes observed after death correspond in the main with those detected during life: the lymphatics are found swollen, the surrounding tissues are œdematous and blood-stained, and in the softened hæmorrhagic glands and local œdema the bacilli are found swarming, sometimes in pure cultures. The entire lymphatic system is found to be involved in the septicæmic form, and the bronchial glands deeply invaded in the pneumonic cases.

Hæmorrhages, when not observable during life in the skin, may be found in all the serous membranes and throughout the entire gastro-intestinal tract after death. The spleen, liver, kidneys, and lungs are the seat of deep congestion and often of hæmorrhagic deposits, and the bacilli are found in all the organs and in the blood. In this latter site the bacillus cannot usually be detected during life, unless the blood be examined a short time before death; but in the septicæmic type the peripheral blood may show bacilli from the onset. Hunter maintains that the bacilli can often be found

at a very early stage of the disease in all the types of cases, but Stephens denies the accuracy of his methods.

Diagnosis.—The disease can be recognised with certainty by an examination of a few drops of the liquid effusion obtained on puncturing the bubo with a hypodermic needle attached to an ordinary hypodermic syringe. The plague-bacillus is easily obtained from the peripheral blood in septicæmic and from the sputum in pneumonic cases. Pure cultures of the organism will produce death usually within twenty-four hours when injected into the peritoneal cavity of a guinea-pig.

Once the advent of an epidemic of bubonic plague has been realised, the diagnosis by signs and symptoms is easy without resort to bacterial investigations. From ordinary lobar *pneumonia* the gravity of the constitutional symptoms from the very onset affords an obvious means of differentiating the disease; these are always out of proportion to the lung-signs. In all doubtful cases the examination of the sputum is conclusive.

Pestis minor or *ambulans* may be mistaken for *syphilis* or *venereal disease*, or any septic infection which causes buboes in the groin or other lymphatic glands; but a careful inspection of the possible sites of inoculation and the history of the case will usually easily establish the differential diagnosis. *Cerebro-spinal* fever may simulate the disease, but the presence of the diplococcus intracellularis in the fluid obtained by lumbar puncture, and the marked retraction of the head, never occur in plague. *Puerperal fever* closely resembles the septicæmic type of plague; but this disease follows delivery or abortion, whilst in plague, in addition to the discovery of the specific bacillus in the peripheral blood, the symptoms precede the emptying of the contents of the uterus.

Typhoid fever, *influenza*, and *small-pox* are differentiated sometimes with difficulty until after the expiration of three or four days; but the gravity of the constitutional symptoms and the demonstration of the plague-bacillus in a drop of fluid from a suspected glandular swelling or in the blood is conclusive.

Prognosis.—The death-rate is variable, being exceedingly high in children and old subjects and in the dark-skinned races, amongst whom the mortality has in some epidemics reached 90 per cent. When the bubonic swellings are situated in the cervical region the outlook is much more serious than when the glands in the groin are affected. The septicæmic type is nearly always, and the pneumonic form scarcely less, fatal. Pregnancy, when it exists, usually renders death inevitable once plague-symptoms supervene. In any instance of the disease the discovery of the bacillus in the peripheral blood justifies a fatal prognosis. In all examples, even of the simple bubonic type, the condition of the heart is a most

important consideration, though the patient may have survived the first eight days, after which, in most cases, the prospect of recovery considerably brightens. It is difficult to estimate the average mortality of the disease: Low places it at 35 per cent. in Europeans treated in hospital; but it varies considerably in different epidemics and at different periods in the same epidemic, being usually much lower at the commencement and towards the end of the outbreak. It is always greatly reduced in those individuals who have been previously injected by Haffkine's prophylactic, and the serum-treatment of Yersin and Lustig appears to afford a means of improving the patient's chances of recovery when the disease is not of the fulminans type.

Treatment.—*Prophylactic* measures are of vital importance in the face of a threatening invasion through plague-stricken ships. Every known means of improving the sanitary condition of the seaport exposed to such a danger should be immediately initiated, and the most rigid isolation of all affected individuals, the destruction of clothing and beds, and the segregation of all persons carrying out these operations should be provided for. The chief agent in the spread of the pestilence being now recognised to be the rat, all sanitary authorities are agreed that the most vigorous campaign against these rodents should be prosecuted. The disinfection of all ships coming from plague-stricken ports should be insisted upon, but unfortunately this can only be thoroughly accomplished when the vessel is emptied of its cargo. The Clayton method of fumigation, by pumping air charged with the fumes of burning sulphur into the hold of the vessel by a modification of the plenum system of ventilation, affords the most efficient means of destroying these pests. Guards should also be used whereby rats can be prevented from reaching the docks by creeping along the mooring-ropes, and the vessel should be kept at a considerable distance from the landing-stage.

A vigorous destruction of all the land-rats should be attempted at the same time, and for this purpose Danysz's virus has proved of considerable value. The immediate cremation or deep burial of all dead bodies and the prolonged isolation of convalescents should be scrupulously superintended by the health-authorities.

Preventive inoculation by Haffkine's prophylactic has given most unmistakable evidence of its worth. The injection consists of the toxins produced by the plague-bacillus, when cultivated in broth for a period of six weeks, along with the dead or sterilised germs remaining in the culture-fluid. The dose being calculated for each batch of the prophylactic after its incubation has been completed, it is injected, like the ordinary antitoxic serums, into the subcutaneous tissue.

Slight inflammatory fever, malaise, and local irritation follow, which usually subside after twenty-four hours. Though the immunity is not always absolute or specific, it affords strong protection against the disease after a period of six to ten days from the injection, the immunity or protection lasting for about as many months. The protective power of the agent is much enhanced by a second injection given ten days after the first dose. It is claimed for the prophylactic that it is sometimes curative when injected in the incubation-stage of plague-disease; but this is doubtful, since the period of incubation is known to rarely exceed five days, and may be as short as two days. It is, however, placed beyond doubt that but a small proportion of injected individuals catch the disease, and the mortality amongst those who do is very trifling.

The sera of Yersin, Lustig, and Bandi, like the anti-diphtheritic serum, are obtained by immunising animals with injections of the bacilli or their toxins, and have been employed as protective or immunising agents; but they afford no advantages over Haffkine's prophylactic. The same remark applies to the injections of dead cultures manufactured by the methods of Calmette and Kitasato.

The treatment of the established disease resolves itself into the relief of symptoms as they arise by the exhibition of remedies employed upon the usually accepted therapeutic lines, fever, when severe, being reduced by cold packs or sponging, and cardiac weakness combated by free stimulation. Hunter, of Hong-Kong, maintains that all cases of plague show an early and extreme cardiac weakness; hence strychnine in full hypodermic doses should be frequently administered from the onset. The local management of the buboes should be carried out upon generally accepted principles; it does not appear that any benefits accrue from the injection of anti-septics. Ice or warm poultices are variously recommended; but there should be no hesitation in free incision and rigid local antiseptic treatment, once signs of suppuration have appeared.

Serum-therapy in plague is a serious and important question, as from the nature of the disease no other known method holds out any prospect of the malady being successfully dealt with. Yersin's serum from horses immunised by large doses of the living bacilli in pure culture has not maintained its first reputation, but there are good grounds for hoping that an antitoxin will be found which will give results approaching those obtained by the diphtheritic antitoxins. In Oporto Yersin's serum appeared to save many lives. The best method of using it is to inject 50 c.c. immediately into a vein, and twice this amount subcutaneously in ten or twelve hours, which latter dose is to be repeated in twelve hours, making

during the first twenty-four hours 250 c.c. Daily 50 c.c. should be given subcutaneously as long as any fever remains.

Haffkine's prophylactic appears to possess little, if any, *curative* value, and the serum of Lustig has been given subcutaneously in doses of upwards of 70 oz. without much apparent benefit in severe cases.

PLEURISY

Inflammation of the pleural membrane exhibits itself in various types or forms, which have led to attempts at a classification of the disease which often is of little assistance at the bedside.

Thus the division into *idiopathic* or *primary*, as contrasted with *secondary*, pleurisy is of as little value clinically as the division of the disease into the *localised* and *diffused* varieties. The division of the affection into the *acute* and *chronic* forms is open to the serious objection that a pleuritis starting abruptly with a most typically acute onset may develop into an essentially chronic type; and sometimes, though rarely, an example of the most insidious and slowly progressive form may suddenly resolve or take on acute features. The grouping of all pleurisies into the *dry* or *adhesive* form and *pleurisy with effusion* is sometimes useful, but as a scientific classification it breaks down at the bedside, because in most of the cases of so-called dry pleurisy there is some amount of effusion.

A bacterial classification is not of much clinical value, since the same organism may in one patient cause a purulent and in another patient a fibrino-serous exudate; for the same reason there is little to be gained by dividing pleurisies into the *fibrino-serous*, *purulent*, and *hæmorrhagic* forms.

It will therefore be advisable to describe the most common form of the disease without any attempt at classification of the less frequently met-with varieties, which may be best described by contrasting them with this common type.

1. ACUTE FIBRINO-SEROUS PLEURISY,

or Pleurisy with Effusion, as it is sometimes called, is, as implied in its name, an acute inflammation of the pleural membrane, accompanied by the exudation into the sac of a highly albuminous liquid containing fibrin and serum.

Etiology.—Individuals of all ages are liable to pleuritis, from the late months of intra-uterine life to old age; and, though the male sex figures at a higher rate in most hospital-statistics, it does not seem proven that sex makes any marked difference in the incidence of the disease when the question of the chances of exposure or opportunities for infection are considered. Trauma, with or without breach of surface

or fracture of ribs, is a factor mainly of surgical interest. Exposure to cold is undoubtedly an etiological factor of great importance, but the explanation of its influence is surrounded with difficulties. According to some authorities, the exposure is considered as a factor akin to trauma, whilst others regard the reduction of temperature as affording an opportunity for the invasion of the body by various micro-organisms, as seen in the case of acute pneumonia and rheumatic fever. Whilst the fact is unquestionable that acute pleurisy often follows immediately after the body has been exposed to severe cold or wetting, several considerations should not be overlooked. Those who so suffer form but an infinitesimal percentage of those constantly exposed to similar conditions; too often the history of the so-called chill, upon careful investigation, is found to be but the initial symptom of the invasion of the disease, and in the after-course of the attack its primary cause, as the tubercle bacillus or other organism, can often be demonstrated.

The consideration of *tubercle* as an etiological factor in the production of acute pleuritis is a wide and important subject, already discussed from one side under the etiology of phthisis. The interest of the problem lies in the difficulty of deciding whether the pleurisy is the *result* or the *cause* of the tuberculosis. There can be little doubt that both views are correct. In many cases of typically acute pleurisy with effusion, some of which rapidly supervene upon a severe chilling of the surface of the body, the bacilli have been found in the exudate, or tubercles have been demonstrated after death in the pleura, lung, or bronchial glands. In many, if not in most, of these acute cases the obvious conclusion is that the cause of the pleurisy was the tubercle bacillus. The frequency with which pulmonary phthisis or tuberculosis in the thorax follows long after a simple acute pleurisy has resolved, and left the individual in apparently sound health for several years, forces the conclusion that the inflammation of the pleura diminished in some way the vital resistance of the tissue, or, in other words, increased the susceptibility to the tubercle bacillus. In this latter case the original attack of apparently simple or non-tubercular pleuritis must be accepted as the *cause* of the subsequent tuberculosis or phthisis, just as a severe trauma of the testicle or spine is often followed by a localised tubercular disease. The important figures quoted by Osler prove this latter contention, though this distinguished authority appears rather to regard them as evidence that the original pleurisy in these cases had been of tubercular nature.

The following must be recognised as etiological factors—viz. such diseases as acute rheumatism, the specific fevers, influenza, septic affections, renal disease, nearly every pulmonary disease save bronchitis, but especially pneumonia

and inflammatory affections of neighbouring organs, as endo- and pericarditis, the growth of tumours, suppurative conditions of the bronchial glands, and spinal caries. These latter-mentioned conditions, like suppuration originating in the liver, kidney, or other abdominal organ, cause pleuritis by simple extension of the inflammatory process. An acute or sub-acute form of the disease is frequently a terminal event in many cachectic conditions.

The modern tendency is to regard all forms of pleurisy as the result of the irritation of certain microbes or their toxins; hence the explanation of the etiological relationships between the pleural inflammation and tuberculosis, pneumonia, influenza, typhoid fever, measles, scarlatina, and several other diseases already mentioned.

The microbes found in the exudate, and which are regarded by most pathologists as the cause of the pleural inflammation, are chiefly three—viz. the *tubercle bacillus*, the *pneumococcus*, and the *streptococcus*; though numerous others have been identified in a small percentage of cases, as *staphylococcus aureus*, *B. typhosus*, *B. coli*, and *B. diphtheriæ*, and the bacillus of Friedländer. In many cases a dual or multiple infection can be demonstrated.

The majority of so-called *idiopathic* pleurisies are held by many to be the result of the tubercle bacillus, and of the *secondary* pleural inflammation so constant in pneumonia the causative agent is regarded to be the diplococcus lanceolatus. This organism is so frequently found in the purulent exudate of the pleurisy of children that the condition has been described as a primary empyema. It must, however, be always remembered that the diplococcus lanceolatus may cause either a fibrino-serous or a purulent exudation; and the same is certainly true of the tubercle bacillus and occasionally of the streptococcus. The pleurisy caused by the streptococcus and staphylococcus pyogenes aureus is usually purulent, and is nearly always secondary to pneumonia, inflammation, abscesses or gangrene of the lung, or septic inflammations in the thorax or abdomen which spread by continuity; rarely it may be the result of a general blood-infection, as in measles, scarlatina, diphtheria, or erysipelas. It is thus obvious that a bacteriological classification of pleurisy is not of great clinical assistance, since the same microbe may produce a serous or a purulent exudation. The pleurisy of rheumatic fever, and that so often found in cancer of the pleura, are usually sterile. The tubercle bacillus is only detected in the exudate in most cases after a prolonged search, and sometimes its presence is only to be demonstrated by the results of guinea-pig inoculation. The use of tuberculin-injections for diagnosis is barred by the presence of fever, but the new method of

conjunctival instillation introduced by Calmette is a great advance.

Symptoms.—In a typical case of acute pleuritis which ends in effusion the invasion is sudden, as in pneumonia, with a chill, smart fever, a catching pain in the side, accelerated and shallow breathing, with cough, and, in the case of children, a general convulsion, followed often by vomiting. In the majority of cases, however, the invasion is not so abrupt as the above combination of symptoms might suggest, its advent being preceded by a shorter or longer prodromal period of malaise and chilliness. In a small percentage of cases the onset is insidious, the disease being for a time latent, the first symptom which attracts the patient's attention often being breathlessness upon some trifling exertion.

Pneumococcic pleuritis is usually ushered in by a more sudden or abrupt invasion than the pleural inflammation which is caused by the tubercle bacillus or the streptococcus.

The most obvious symptom is the pain. This is of a severe stabbing character, aggravated greatly by inspiration; it is localised usually about the region of the nipple, below the armpit, or above the clavicle. Sometimes it is felt in the abdomen below the sternum, or near the umbilicus, or in either hypochondrium. More frequently, however, the lancinating thoracic pain is well localised, whilst other severe pains radiate along the intercostal nerves of the affected side to regions at a considerable distance. There is usually marked tenderness of the skin, though the patient often grasps the affected side in his outstretched hand in a vain endeavour to fix the thorax so as to diminish the breathing-movements, as these increase his suffering. After effusion takes place the pain usually lessens considerably and is only felt upon deep inspiration or coughing, but it often returns in a minor degree after absorption.

The fever, though often smart, is rarely as high as that observed in acute pneumonia. Thus it is unusual for the thermometer to register above 103° , whilst in rare cases the temperature may remain normal, even when the exudate is purulent; and as a rule there is no close relationship between the degree of fever and the nature of the pleural contents. The duration of the pyrexia and its remitting oscillations are very variable; usually in large effusions the fever continues as long as the effusion remains considerable, the morning temperature registering often about 101° , with a rise of 1° or more in the evening. In many cases it assumes a distinctly hectic or septic type, with profuse perspirations, and occasionally it is accompanied by diarrhoea, with offensive-smelling stools. In mild cases, and where the effusion is moderate, the temperature falls, and may remain normal after the end of the second or third week. In the acute

stage the surface-thermometer may register one or more degrees higher upon the affected than upon the sound side. Profuse sweating is a very prominent symptom in most cases of pleurisy, and must not be accepted as evidence of a purulent secretion.

Cough is sometimes absent throughout, but, as a rule, it is an early and painful symptom, and usually unattended by expectoration or only accompanied by a scanty mucoid or blood-streaked sputum. Severe paroxysmal coughing at a later stage, with profuse purulent secretion, which may be fetid, marks the rupture of an empyema into the bronchial tract.

Dyspnœa is often noticeable from the onset ; as a rule, it is in proportion to the rapidity with which the exudation is poured out rather than to the actual amount of fluid present. Thus in chronic cases, with one lung compressed almost entirely by an accumulation reaching to above the clavicle, there may be no marked frequency in the respiration-rate, unless after some brisk movement ; whilst in the acute disease half this amount of fluid may by the fourth day after the onset cause marked dyspnœa. The dyspnœa of the onset is caused by the involuntary inhibition of breathing, the result of the thoracic pain ; that which occurs at a later stage is due to the compression of the lung and the displacement of organs. It may, however, be the result of pneumothorax or other complication. The blood-condition has been carefully examined in many cases ; usually there is but a slight degree of leucocytosis, and this only in the early stage, but when pus exists the number of white cells may reach above 25,000 per c.mm. of blood.

Clubbing of the fingers is usually present in all chronic cases ; but this sign may show itself also in acute cases within the first fortnight.

Physical Signs.—The earliest sign will be that observed on auscultation, and it is known as the *friction-sound* or *friction-rub*. When present it may be regarded as pathognomonic of the early stage of pleural inflammation ; but it is often absent, and as a rule is not heard while fluid effusion separates the inflamed surfaces of the pleura. It is a sound of a distinctly rubbing or grating character ; in some instances it may be so fine as to be absolutely undistinguishable from the typical pneumonic crepitation, in which case, in the writer's experience, it is heard over a considerable area of the posterior part of the thorax on the affected side. This variety of friction-sound conveys the idea of being produced immediately under the skin, and very closely resembles the phenomenon observed when auscultation is attempted over a part of the chest covered by strong crisp hairs. Between this sound, which seems to be made up of a series of shorter

sounds and the creaking noise resembling that produced by the bending of sole-leather, there are all sorts and degrees of loudness or harshness. All varieties of the friction-rub are superficial, are rendered more obvious by pressing the stethoscope firmly against the chest-wall, and they convey the idea of dryness, save in the instance of the crepitating friction-rub, which suggests stickiness or a greasy condition of the pleural surfaces. The most typical form of the sign in acute pleurisy, however, is that which is heard both in inspiration and expiration, being a to-and-fro sound or rub, audible over the most movable part of the thorax—viz. outside the nipple towards the axilla, and usually markedly localised or circumscribed. When heard near the heart it may be mistaken for a pericardial sound, but it disappears upon suspension of the respiratory movements. The period during which it is audible depends upon the rapidity with which the exudation is poured out, but it may occasionally continue to be heard above the level of the liquid, and it often is again heard for a considerable time after the effused liquid has been absorbed.

The following signs are observable after effusion has taken place :

Inspection will reveal increased movement on the sound side and diminished movement upon the affected side—a sign which is, however, present from the first onset, owing to the pain caused by inspiration. After effusion has occurred, the patient usually lies upon the bad side to give more scope to the play of the muscles of respiration on the sound side. The affected side of the thorax looks to the eye to be considerably enlarged owing to the obliteration of the intercostal depressions, which may in rare cases exhibit distinct bulging. The shoulder is raised and the spine curved towards the sound side. Inspection will also reveal displacement of the heart when the effusion is extensive, the pulsation of the right conus in left effusions being sometimes visible in the right nipple-line, and often epigastric pulsation is evident. In right pleurisy the apex-beat is elevated and pushed towards the left axillary line, and the lower margin of the liver is depressed.

Mensuration may show an increase of nearly two inches in very large effusions ; but as a rule there is only trifling increase demonstrated by the use of the ordinary tape-line. The cyrtometer, however, reveals marked increase in the antero-posterior measurement, as the affected side tends always towards the semicircular form.

Palpation corroborates the evidence obtained by inspection. With great care the finger-tips may appreciate a degree of increased resistance, which affords unmistakable evidence of effusion as each space is delicately palpated from above

downwards or from below upwards, though no fluctuation can be detected. With practice this evidence may even be more reliable than that obtained by the percussion-sounds. The vocal fremitus or thrill is nearly always diminished, and in large effusions entirely absent, over the whole side. A friction-fremitus or thrill may sometimes be felt in the early stage before effusion has taken place, or it may be detected later on after absorption has occurred.

Percussion reveals dulness over the area of the effusion, and the sense of increased resistance already perceptible upon palpation is obvious. The dulness is detected first in the posterior aspect of the lower zone of the affected side, and its increase may be demonstrated from day to day, as the effusion mounts upwards. As a rule, this valuable sign is not unmistakably marked at its upper border unless the layer of liquid approaches towards an inch in its antero-posterior diameter. It is never absolute on deep percussion until the lung becomes compressed and more or less airless. If the patient has been keeping the recumbent posture, usually the greater portion of the back is dull, whilst the anterior region is clear, or almost so. In moderate effusions the chest-wall often gives a tympanitic note above the level of the fluid: this sign is known as Skodaic resonance; sometimes, though not often, a distinct cracked-pot sound may be elicited below the clavicle.

The upper limit of dulness is not horizontal with the patient in the erect position, light percussion revealing a dome-shaped outline, with the apex usually in the region of the axilla. A very important feature in the dulness of pleural effusion is that it does not shift or alter its outlines obviously upon changes of posture as does the dulness of ascitic fluid. It is, however, an error to say that no change can be detected by this manœuvre. Though a bed-ridden patient with a moderate effusion may only exhibit dulness posteriorly upon assuming the erect posture, if he be made to breathe deeply for several minutes, and the examination be repeated, there is usually a marked dulness observable after some time over the front of the chest. The opposed pleural surfaces hold up the fluid between them for a considerable time after a change of posture because of the collapse of the lung, but ultimately the force of gravity, combined with deep breathing, tells. Shifting dulness probably never occurs in purulent exudations.

The quality of the dulness is somewhat characteristic; it amounts to absolute deadness when the lung is completely collapsed. The description of the dulness being of a wooden character is given by those who probably confuse the acoustic phenomenon with the increased resistance felt by the finger in the act of percussion.

The extension of dulness beyond the normal limits upon the sound side will afford evidence of the degree of the displacement of organs. When the sac is completely full on one side the mediastinum is displaced, so that dulness may be elicited beyond the sternal margin of the sound side, and there may be entire absence of resonance over the apex of the distended sac above the clavicle. A triangular patch of dulness may often be demonstrated on the sound side behind, with the apex about the level of the angle of the scapula.

Percussion and palpation of the abdomen will reveal the displacement of the liver, spleen, and stomach. Sometimes the percussion-signs are modified by old adhesions, though encysted effusions are not commonly met with. It must be always remembered that a high degree of pleural thickening will yield a comparatively dull sound on moderately light percussion, and it is not uncommon for considerable dulness to remain long after the absorption of the fluid, especially when the side retracts and the lung fails to expand owing to extensive adhesions.

Auscultation.—The friction-rub already described is absent when effusion takes place, but sometimes it may be still heard above the level of the liquid. It reappears for a short time after the absorption of the effusion permits the fibrin-covered surfaces to come into contact again—a phenomenon comparable with the redux crepitation heard in pneumonia.

There is usually marked diminution or entire absence of vocal resonance over the region in which the vocal fremitus has disappeared, corresponding with the absence of vocal fremitus detected by palpation.

The breath-sounds are usually weakened, the vesicular quality being discernible with difficulty, and often the sounds are entirely suppressed; but the opposite condition is not uncommon, and introduces a problem of great complexity into the diagnosis. Thus, especially in children, the breathing may be found to be *bronchial* in character all over the affected side; this is probably due to the patency of the large and medium-sized bronchi. Under these conditions the vocal resonance is increased, and often about the angle of the scapula bronchophony is well marked; sometimes this takes on the peculiar bleating quality described as *ægophony*, especially when the effusion is moderate and the resonance clear in front above the level of the liquid.

Bacelli's sign is believed by many to be distinctive of a purulent exudation; this observer maintained that the whispered voice is heard distinctly through a fibrino-serous effusion, but not through a purulent one.

In many cases, where the breath-sounds are distinctly intensified so that the bronchial character of the breathing is unmistakable, the steady filling of the entire sac will cause

their complete obliteration as the dulness becomes absolute from the base to the clavicle ; but occasionally the bronchial character of the breathing and the intensification of the voice-sound may remain after the pleural sac has been distended. In this latter condition, liable to be mistaken for pneumonic consolidation, Gee points out that percussion in front by means of two coins will give to the ear applied to the back of the chest a clear bell-like or metallic sound. Auscultation will also enable the examiner to corroborate the degree of displacement of the heart, especially when the apex-beat is not visible ; sometimes a systolic pulmonary murmur may be audible, and a pleuro-pericardial rub may be detected. As might be expected, the breath-sounds are intensified upon the sound side, the breathing usually assuming the typical puerile type.

Puncture gives the only unmistakable evidence of fluid, and should not be omitted. As a rule, the ordinary hypodermic needle and syringe are quite reliable. Very rarely will the aspirator-needle give a positive result after failure with a good air-tight hypodermic apparatus ; but certain precautions are essential to success. The puncture may be made in almost any situation : the lateral region of the chest between the angle of the scapula and the axillary line is probably preferable ; it is a common mistake to insert the needle too near to the lower margin of the thorax. Antiseptic precautions are obviously imperative, and the syringe should contain a few minims of a sterile saline-solution, so that after the insertion of the needle its calibre may be cleared by pressing the piston home before suction is attempted. If ordinary antiseptic solutions—mercurial or carbolic—are used, these will cause coagulation of the albumin in the serous exudate and may lead to the erroneous conclusion that pus is present.

A microscopic examination of the fluid will sometimes reveal the specific organisms already mentioned under Etiology, and will afford valuable indications for prognosis and treatment.

Under 'Empyema' the signs and symptoms of a purulent effusion have been already detailed ; but, as obvious from the above description, these are in the main identical with the clinical picture of the fibrino-serous form.

Course and Duration.—These are exceedingly variable. Usually, even in severe cases, after the expiration of a week or two the fever and constitutional disturbances show signs of abatement, and evidence of absorption having already set in is forthcoming. Thus the displacement of organs caused by a large effusion diminishes steadily in degree, the diaphragm rises, the heart swings round towards its normal position, the liver, spleen, and stomach resuming their proper sites. At this stage auscultation may detect the redux

friction-rub as the distension of the affected side diminishes; the vesicular quality of the breath-sounds becomes audible, vocal fremitus and resonance return, and the dulness steadily lessens, though this may long remain, and in a small percentage of cases extensive basal dulness may be a permanent feature. Extensive effusions may disappear completely within a week after the first turn of the tide has manifested itself, the dulness from day to day showing a perceptible falling downwards towards the lower thoracic margin, though in rare cases the liquid seems to begin to disappear from all parts of the pleural sac at the same time; but always some lingers in the lowest portion of the cavity for a considerable period.

Exudations which have been of slow and insidious onset are usually much more tardy in their disappearance than are the more acute effusions; and in the writer's experience this is true of the tubercular cases especially. Many of these latter drag on for several months if not treated by aspiration.

Some degree of retraction of the side is the rule, and this invariably follows when the carnified lung, bound down by dense adhesions, is prevented from expanding. In this latter case the condition is permanent, the mediastinum being dragged or pushed over to the affected side, and all the organs—heart, liver, spleen, &c.—remain displaced and the spine twisted. Permanent retraction of marked degree is a more common event after purulent than after fibrino-serous effusions.

ACUTE FIBRINOUS, DRY, OR ADHESIVE PLEURISY

In this condition, as implied in its title, no perceptible effusion of liquid occurs. The inflamed pleural surfaces are covered over with a layer or a series of layers of solid lymph, causing symptoms more or less closely resembling those observed in the first stage of pleurisy with effusion—*i.e.* pain in the side, some pyrexia, cough, and even dyspnoea. As a rule, however, the symptoms are much less acute than in the cases where effusion is abundant, and all signs may be absent save the friction-rub and the diminished movement of the affected side of the thorax. It must not, however, be inferred that because marked friction is audible over a considerable area of the chest-wall there is no fluid present in cases which seem to be essentially of the dry variety, as puncture of the lower part of the sac will usually give some fluid.

Often a well-marked friction-fremitus may be appreciated by the hand as the patient takes a deep inspiration.

The causes of dry or adhesive pleurisy are mainly two. In a very considerable percentage of cases a history of chill or exposure is obtainable. The frequency with which extensive pleural adhesions are found in the *post-mortem* room

would seem to support the assumption that the condition must be commonly caused by some temporary alteration of the normal functions of the pleura independent of the admission of tubercle, streptococci, or pneumococcic organisms. The second group of causes include those primary diseases of the lung which induce a secondary pleural inflammation of a localised character. This is witnessed in tubercular disease of the apical region, where phthisis is almost constantly found associated with secondary pleuritis. In the same manner acute lobar pneumonia is very commonly associated with more or less solid or fibrinous exudation on the surface of the pleura covering the affected lobe, and a similar condition accompanies any other inflammation or neoplastic growth involving a portion of the pulmonary organ.

CHRONIC PLEURISY

This may be a sequence of an acute attack, either with or without liquid effusion, so that both chronic pleurisy with effusion and chronic dry or adhesive pleurisy are often met with. The former is sometimes seen to follow a typical acute case, in which after aspiration the fluid reaccumulates, necessitating frequent tapplings; the ultimate issue generally demonstrates the tubercular nature of these cases. In another variety of this chronic form, usually designated *latent pleurisy*, the physician is consulted by a patient for 'shortness of breath' without any constitutional disturbance of any kind, and upon examination one pleural sac is found to be distended with fluid from the clavicle to the diaphragm, exhibiting all the *physical signs* without the symptoms already described as characteristic of the acute variety of pleurisy with effusion. Many of these cases after tapping clear up and remain well for many years, save that there is generally some degree of crippling of the respiratory powers from deficient expansion of the lung; but a considerable percentage of them end in phthisis. It is doubtful whether such cases should be considered as of primary tubercular origin or should be regarded as examples of tuberculosis supervening in an individual whose natural resistance has been lowered by a previous chronic inflammation.

Chronic dry pleurisy may remain as the sequel to an acute attack in which there has been originally a more or less extensive fluid effusion. This result is more liable to follow a purulent effusion, and is seen in its most typical form in those cases of empyema where perforation of a bronchus has occurred and led to the complete evacuation of the pus. The lymph upon the opposing pleural surfaces becomes slowly organised, and the gradual shrinking of the newly formed cicatricial tissue causes a progressive retraction of the affected side, commencing in the basal region and ultimately pre-

venting the expansion of the lung, which remains airless and carnified. Even when this latter result has not followed, the proliferation of the connective-tissue elements may possibly extend to the pulmonary tissue, eventuating in cirrhotic changes and causing bronchiectasis—a condition akin to the so-called ‘fibroid phthisis.’

The dry secondary pleuritis of tubercle is usually a localised chronic process, but occasionally it may assume an active character and spread over a considerable area of the sac, leading to enormous thickening of the pleural membranes. Osler describes a dry *primitive* chronic pleurisy of latent type. This may be regarded as analogous to the ‘silent’ or ‘latent’ form in which extensive effusion occurs without symptoms, except the breathlessness observable upon physical exertion.

EMPHYEMA

This has already been dealt with under its own title. For convenience of reference a brief notice is here required, as very occasionally the condition supervenes upon a pleurisy which has apparently commenced as a simple fibrino-serous inflammation.

Pus in the pleura may be the result of infection by the pneumococcus, staphylococcus, streptococcus, or other pyogenic organism, by the bacillus of tubercle, and occasionally by the influenza bacillus. It is a common form of pleurisy occurring in association with scarlatina, influenza, measles, typhoid fever, acute pneumonia, and occasionally in connection with septicæmia, chest-wounds, disease of bronchial glands, spinal caries, &c.

The symptoms and signs are, in the main, those of an acute pleurisy with effusion. It cannot be too strongly emphasised that the degree of constitutional disturbance affords no criterion of the nature of the effusion in pleurisy. The fever and sweating may be severe and even excessive in simple fibrino-serous cases, and, with the pleural cavity full of pus, there may be little pyrexia and no hectic symptoms. Moreover, a purulent effusion often is marked by the most insidious and latent form of onset, without pain, cough, or sweating. Leucocytosis, when well marked, is, however, strongly suggestive of empyema, and, as a rule, the displacement of organs is more obvious than in the serous form, especially in the case of children, in whom the disease is more frequently met with than in adults. The only method, therefore, of recognising the condition with any degree of certainty is that of puncture or aspiration; and it should be remembered that, owing to the greater degree of thickening of the pleural membrane and to the fact that the pus is often loculated, a difficulty may be experienced in striking upon the exudate.

The fluid of empyema may exhibit all degrees of opalescence from turbidity to thick gelatinous or creamy pus. It may be 'laudable' or indescribably fetid or gangrenous; the latter condition has been repeatedly observed by the writer in the empyema following influenza.

When the effusion of pleurisy is seen to pulsate with the cardiac rhythm the fluid may be almost certainly diagnosed to be purulent; and though very occasionally a fibrino-serous pleurisy has been known to point at some portion of the chest-wall, the appearance of a tumour may be accepted as practically a positive proof of pus. This condition, known as *empyema necessitatis*, may or may not be accompanied by pulsation. When the pus ulcerates into the lung and is expectorated by the bronchial route, or when it perforates the œsophagus or finds its way into the pericardium, stomach, intestine, peritoneum, or appears as a psoas or subphrenic abscess, the nature of the case is generally obvious.

Morbid Anatomy of Acute Pleuritis.—The usual changes found in inflammatory serous affections are observed. The membrane at first is congested, loses its polish from the desquamation of its endothelium, becomes thickened by proliferation of its connective-tissue elements, and usually is covered over by membranous layers of fibrinous deposit. In the later stages the opposed surfaces of the pleura become more or less adherent, and all degrees of adhesions from thready or ribbon-like bands to firm general gluing together of the two layers and complete obliteration of more or less of the sac, may be found.

The fluid found in the pleural cavity varies considerably in appearance owing to the presence of blood, pus, and inflammatory products of different kinds. In ordinary fibrino-serous exudations it resembles the liquor sanguinis, is of a lemon or greenish-yellow colour, varying in s.g. from 1005 to 1030. It usually coagulates spontaneously when left to cool in a vessel outside the body, and always coagulates immediately upon the application of heat owing to its richness in albumin. Urea, sugar, cholesterin, and peptone are found along with chlorides. There is a variable amount of fibrin present, which often renders it opalescent if in the form of fine shreds; but white curd-like flakes, especially in the fluid removed from the lower portions of the sac, are commonly present, and may even exist in considerable masses.

Under the microscope various types of white cells, blood-corpuscles, fibrin-flakes, and pus may be found, in addition to the specific organisms already mentioned. Much importance has been attached to the kind of leucocytes present in the exudate, but there is considerable difficulty in arriving at an accurate conclusion of the cause of the pleurisy by a mere cytological diagnosis.

The subject is of great importance owing to the fact that so large a percentage of fibrino-serous pleurisies are tubercular, and especially as there is almost always great difficulty in detecting the bacillus of tubercle in the effusion. Sometimes also *inoculation* of guinea-pigs fails to demonstrate the tubercular nature of the exudate; *tuberculin-injections* administered to the pleuritic patient are only admissible in the apyrexial cases, and, as pointed out by Hadley, *cultivation* of the fluid will not show colonies till almost after the expiration of a month; and the *agglutination-test*, unless in the hands of an expert, is of little value.

Hence the desirability of any means by which a conclusion can be arrived at regarding the nature of the primary cause in apparently sterile fibrino-serous pleurisy; and although the subject rather belongs to the section of diagnosis, it is better discussed in connection with the morbid anatomy of the disease.

Dieulafoy finds that much reliable information is obtainable by a study of the preponderating type of leucocyte present in the effusion. He states that in tubercular cases the lymphocyte or small mononuclear form is found in greatest abundance, whilst in the pneumococcic and streptococcic infections the polymorphonuclear greatly preponderate. Though Gibson and Longcope regard these results as evidence of the degree or intensity of the inflammation, and not of its primary cause, nevertheless the method promises to be of great value, since the tubercular organism is as a general rule much milder in its inflammatory action than the pneumococcic and streptococcic organisms, which cause severer inflammation, characterised by the outpouring of abundant polymorphonuclear cells.

Hadley recommends Jousset's method of examining the *débris* of the digested clot after spontaneous coagulation of the exudate. (See page 1026.)

In the effusions of asthenic patients suffering from pleurisy there may be much blood present; but as a rule the so-called 'hæmorrhagic' pleurisy is an indication that the disease is either of a malignant nature or of tubercular origin, when the absence of the purpuric state can be demonstrated.

The amount of fluid found in the pleura in acute pleurisy varies from a few drachms to half a gallon or more; generally speaking, somewhere about three pints may be accepted as an ordinary average.

The displacements of organs observed during life are found after death; the heart is seen to be pushed usually as a whole towards the sound side by displacement of the mediastinum, the apex being but slightly, if at all, rotated. In left-sided effusion the part of the heart found in contact with the chest-wall is made up chiefly of the right ventricle.

Rarely is any kinking of the great vessels visible. The lung is found collapsed, often quite airless, and compressed in all its lobes; and this condition, it must be remembered, may exist when the amount of exudate is not very large. Sometimes only the lower lobe is seen to be compressed and atelectatic. The compression of the lung found in pleurisy with effusion is not capable of the apparently easy explanation that it is the simple result of the pressure of the exudation, because this is much lower than is usually realised. Rosenbach insists that the compression is the result of changes which materially interfere with the normal *tonus* of the organ.

Over the compressed and airless lung the fibrinous exudation may be so solid, and its layers so numerous, that it may be difficult or impossible to inflate it.

OTHER VARIETIES OF PLEURISY.—In *children*, acute pleurisy, as might be expected, runs a more rapid course, is more frequently found to be purulent, is usually caused by pneumococcic infection, and is more amenable to treatment. The onset is more abrupt than in the adult, being often ushered in with a severe convulsion, though latent pleurisy in children is also occasionally to be met with. The physical signs correspond to those observed in adults, save that, owing to the more yielding character of the chest-wall, bulging may be very obvious, and generally the bronchial type of breathing is present. It is this latter fact which often causes the pleurisy of childhood to be mistaken for pneumonia, especially as there is less displacement of the heart, and the intensified breath-sounds may be heard all over the affected side and friction-sound is comparatively rare at any stage. Puncture affords the only reliable method of diagnosis in the acute pleurisy of childhood. According to Netter, purulent pleurisy in children often follows an attack of pneumonia, this form, known as *meta-pneumonic empyema*, being always caused by the pneumococcus.

Encysted Pleurisy.—In this form the fluid is circumscribed by adhesions which have previously existed or which have formed during the attack when this has pursued a slower course than usual. The fluid may be confined in one pouch, but more frequently two or more loculi are present. The physical signs correspond, the dulness being distinctly localised; but the diagnosis is puzzling till cleared up by puncture, which usually reveals pus, since these localised accumulations are more frequently observed in empyema than in fibrinous pleurisies. The writer has observed this condition to follow an attack of typical lobar pneumonia upon two occasions in adults. The diagnosis may be impossible during life when the pouch of pus is located between the base of the lung and the upper surface of the diaphragm. The symptoms

in these cases of encysted empyemata are practically identical with those of a pulmonary abscess after the pus has perforated the lung-substance and been expectorated.

Diaphragmatic Pleurisy.—This form, as already stated, closely simulates abdominal inflammation. The usual *symptoms* of acute pleuritis are present, but the pain is usually more intense and the difficulty of breathing from the onset is much more formidable and alarming, cyanosis and anginiform attacks being not unusual. The progress of the case is, however, more satisfactory than the abrupt and severe onset would suggest, since in the great majority of instances the type of inflammation is usually plastic or dry. The relief afforded by a hypodermic injection of morphia is a valuable aid in the diagnosis, unless in the rare cases where pus has formed. *Signs* are obscure or entirely absent, save those which are produced by the inhibition of the respiratory movements, and these are to a large extent dissipated by the relief afforded through the narcotic.

Interlobar Pleurisy.—This in slight degree is a common event in all acute pleurisies. When fluid is poured out between the pleural reflection between the lobes of the lung and circumscribed by adhesions, the physical signs may be almost nil, since the effusion does not usually come into direct contact with the chest-wall. The condition is, however, of no clinical importance, save when the loculus contains pus. Under this latter condition, which is not very rare in the secondary pleurisy of tuberculosis and acute pneumonia occurring in debilitated individuals, the clinical picture becomes practically identical with that of a pulmonary abscess. The pus may finally burrow into the lung and be expectorated through a bronchus, or a pyopneumothorax may develop.

Tubercular Pleurisy.—This has been fully dealt with in the preceding description of the disease, and need not be again referred to, save to remind the reader (1) that a large percentage of so-called acute and chronic idiopathic pleurisies are of tubercular origin—indeed, some authorities have been led to regard all cases of acute pleurisy as tubercular. That this latter theory is incorrect is highly probable when we consider that (2) all cases of pleurisy, however caused, are liable after the lapse of a long period—often of several years' duration—to be followed by pulmonary tuberculosis, owing probably to diminished tissue-resistance caused by the original non-tubercular inflammation; and there are, as already stated, good grounds for regarding the tuberculosis as the *result* and not as the *cause* of the original pleurisy.

Hæmorrhagic Pleurisy.—The presence of a bloody exudate has led some authorities to describe this condition as a separate variety of pleurisy, but the appearance of blood in the fibrino-

serous effusion, in the majority of instances, should be regarded as an accidental occurrence. It is much more liable to occur in carcinoma and tubercle, the escape of the red cells being due to the rupture of newly formed blood-vessels; but it is not a rare occurrence in the terminal pleurisy seen in Bright's disease, purpura, cirrhosis of the liver, and other profound cachectic conditions. Extensive hæmorrhage into the pleural sac is also not rarely caused by puncture of an artery in the thoracic wall or by injury to the lung in the operation of aspiration.

Purulent Pleurisy.—This has been already dealt with in the description of the acute affection (p. 1383), and for convenience of reference it is also described under its own title in the article on 'Empyema' (p. 369).

Chylous Pleurisy, or Chylothorax, is so rare as to be but of pathological interest. It is caused by some injury or rupture following disease of the thoracic duct or its branches.

COMPLICATIONS OF PLEURISY.—Pericarditis is not an uncommon complication in left-sided effusions; but it may also occur when the right sac is the seat of pleurisy, the effusion being sometimes purulent.

Peritonitis is a rare complication and probably only occurs in streptococcic infections, in which cases cerebral abscess or meningitis may also result.

Lung-complications are more apt to occur, as collapse, fibroid induration, gangrene, and perforation, the latter leading to pneumothorax or pyopneumothorax.

Thrombosis of the femoral vein is a not uncommon complication; the writer has witnessed it often in mild pleurisies apparently caused by exposure to cold.

Albuminuria is often present, especially in empyema, after the evacuation of the sac; at a later stage it may be a sign of amyloid degeneration following prolonged suppuration.

Paralysis of the vocal cords is a rare complication. However, it is worth remembering that it does occur, probably owing to the pressure of a fibrinous exudate on the left side.

Dilatation of the heart, with general anasarca, is a not uncommon event in cases where the carnified lung remains bound down by dense adhesions.

Considering the large percentage of tubercular cases, it is noteworthy that acute miliary tuberculosis is a rare complication, but it does sometimes occur. Occasionally inequality of the pupils has been observed; this may be the result of pressure of the exudate upon the sympathetic, but it is more likely to be a reflex caused by peripheral irritation.

Diagnosis.—The physical signs already detailed afford, in the majority of cases, almost certain evidence of fluid when this exists in large quantity. The absence of breath-

sounds and vocal fremitus, the presence of a marked degree of dulness on percussion, and especially the evidence of displacement of the heart, seldom leave any doubt about the nature of the case. Puncture, with the precautions already mentioned, should never be omitted when a shadow of doubt exists; only in the merest fraction of exudation-cases will a negative result be obtained if sufficient care is taken. By withdrawing the needle rapidly, without permitting the piston to descend, a single drop of pus may be detected at its point, which is sufficient to establish the diagnosis even when the operation appears to give a negative result. Much valuable information may be obtained by estimating the amount of resistance which the needle experiences at different depths, and by noting whether or not its point moves freely in the pleural cavity before suction is resorted to. The importance of an early resort to puncture is obvious in purulent cases, and a negative result should never be accepted till the operation has been performed by aspirating at different depths from the surface, since often in empyema the pleura is greatly thickened; and when failure still follows this plan, a needle of larger calibre should be substituted for the ordinary fine one. The surgeon should never commence operative procedure at any spot in the thorax without first satisfying himself about the presence of pus below the site which he has selected for his incision.

The following conditions must be carefully differentiated from moderate pleural effusions:

Pneumonia is recognised by its more acute and abrupt onset with a higher degree of fever than is usually observed in pleurisy; the characteristic rusty sputum, when present, is highly characteristic. It must, however, be remembered, especially in the case of children, that pneumococcic pleurisy may possess a similar onset, being sometimes ushered in with a smart rigor or convulsion. In these latter cases the dulness may for a considerable time be confined to the base, and bronchophony may be well marked. A differential diagnosis is only impossible in such a case when a carefully made puncture gives a negative result. Again, in some examples of pneumonia, owing to obstruction of a main bronchus, vocal fremitus and vocal resonance and the breath-sounds may all suffer diminution, a pleural rub may be audible, and friction-fremitus may be felt; but the danger of regarding such cases as pleuritic is slight if repeated examinations be made after a fit of coughing which clears the obstructed air-vessel. A very slowly resolving pneumonia occasionally presents the acme of difficulty when a little fluid exudation lying in the bottom of the sac accompanies the solidification of the lung, and when a friction-rub is audible above the level of dulness.

Pericardial effusion, when extensive, may closely simulate a left pleurisy, especially when a considerable degree of collapse of the left lung is produced by the pericardial pressure. The dull area is triangular or pear-shaped, with the base downwards. The heart is not pushed towards the right, as in pleural effusion: its apex may be often felt in the normal position or slightly raised, and there is always muffling of the heart-sounds; this may be so marked as to render the sounds inaudible. The pulmonic second sound is usually highly accentuated, Skodaic resonance is not heard above the level of dulness, and the lowest part of the lung behind will usually give a resonant note.

In pericardial effusion of moderate degree, in which a pericardial rub is audible, this latter must not be confused with the pleural friction-sound. It is a very characteristic to-and-fro sound, heard only over the heart and not suppressed by cessation of the breathing.

Malignant disease of the lung may closely simulate pleural effusion, especially when of rapid growth, in which case it may cause displacement of organs. Moreover, as it is almost certain to have associated with it some serous effusion, the physical signs may be identical, even to the extent of puncture-results. However, there is never any difficulty in such cases unless the growth starts in the lowest part of the lung, and even then some area of pulmonary tissue, escaping in the general infiltration, will give auscultatory evidence of the entrance of air. The history and progress of the case are usually sufficiently clear to settle the diagnosis, which is rendered positive by the appearance of secondary deposits in the glands or other organs. The same remarks apply to other tumours of a malignant nature arising anywhere within the thoracic cavity. The presence of hæmoptysis is strongly in favour of malignancy.

Collapse of the lung, which is a common complication of the capillary bronchitis of children, may cause a large dull area over the base, which might be mistaken for a pleural effusion. The main points in the differential diagnosis are the evidence of the original bronchial trouble in both sides of the chest, the history of the bronchial onset, and the absence of bulging, which is usually well marked in the pleural effusions of childhood. Collapse of a large portion of the lung will often cause an immediate retraction of the chest-wall over it, and there is no obvious displacement of organs.

Basal phthisis and *Fibroid induration* or *Cirrhosis* of the lung, when not differentiated from a pleural effusion of moderate degree by their chronic progress and history, will be recognised by the negative results of one or more punctures or by the recognition of the bacillus in the sputum. They are, however, only likely to be mistaken for the retracted condition

of the thoracic wall, which sometimes persists long after the absorption of effusion.

Hydatid cysts arising within the thorax or projecting from the dome of the liver into the right pleura may afford physical signs almost identical with those of a pleural effusion; but puncture will at once demonstrate the nature of the hydatid liquid.

Abscess of the liver and *Subphrenic abscess* are often mistaken for a right-sided purulent effusion. The history of the case is of great importance, as careful investigation will seldom fail to detect the presence of some primary malady, as cancer or ulcer of the stomach, hepatitis, hydatids, pancreatic disease, or gall-stones, &c. A case of subphrenic abscess in which air is present in the sac closely resembles a pyopneumothorax; the majority of such are the result of perforation of a gastric or duodenal ulcer.

Actinomyces of the lung, when the disease is situated at the base, may sometimes closely simulate pleural effusion, especially as there is nearly always some fluid poured out into the pleural cavity by the irritation of the fungus; and occasionally the case when first seen may present the typical features of an *empyema necessitatis*. The discovery of the characteristic ray-fungus in the sputum or discharge will easily settle the diagnosis.

Hydrothorax is usually easily differentiated, since it almost invariably is double, though often there is more liquid in one pleural cavity than in the other. In the rare cases reported in hepatic and cardiac conditions, where the transudation is confined to one pleural sac, the physical signs and symptoms of the primary malady are always obvious. Dyspnoea, high fever, and rapid onset characterise a double acute pleurisy, which is, upon the whole, a rare condition.

Rosenbach and Pohl differentiate a passive transudation from an inflammatory exudate by the administration of iodides by the mouth, iodine being speedily found in the hydrothorax fluid by adding fuming nitric acid and agitating with chloroform. In pleuritic fluid a negative result is obtained.

Encysted effusions may lead to serious errors in diagnosis, though they are much less frequent than is usually supposed. They occur in acute cases, especially between the lobes of the lung; the fluid may be encysted or loculated by the presence of former old firm adhesions over any part of the pleural surface. Deep puncture performed with due care will usually demonstrate the nature of such cases.

Abscess in the thoracic wall must be carefully differentiated from a pointing empyema. The well-marked indurated and raised border of a local thoracic-wall abscess, with its great tenderness and vivid red blush, is very seldom, if ever, seen in

empyema necessitatis, and the pus in abscess has always a higher s.g. than that found in empyema.

The diagnosis of dry pleurisy is almost always easy: the characteristic friction-rub can seldom be mistaken for anything else. The rare variety of friction-sound which is described as due to greasiness of the pleura sometimes cannot be distinguished from the moderately fine crepitation heard in bronchitis, and it may closely simulate the finer crepitant râle of pneumonia. This sound is believed by Gee to be a true *bronchial râle*, and not a soft pleural rub; and he depends upon its unilateral distribution as the only means of diagnosing the pleuritic inflammation with which it is accompanied from acute bronchitis.

Pleurodynia can be differentiated from dry pleurisy by the absence of the characteristic friction-sound. Fever, when present, tells in favour of the pain being of pleuritic origin; but in the class of case in which the difficulty arises, unfortunately, fever is generally absent. Diaphragmatic pleurisy is often adhesive, and may be easily mistaken for abdominal disease; the intensification of the pain and dyspnoea upon deep inspiration is, however, characteristic, as are also the intensity of the pulmonary embarrassment and the relief afforded by a dose of morphia, which permits the breathing to be deepened.

The diagnosis of the nature of the primary cause—pneumococcic, streptococcic, or tubercular—has been already fully dealt with in the description of the effusion upon page 1374.

Prognosis.—The immediate outlook is good; few deaths occur from the direct results of the pleural inflammation or from the pressure of the exudation. The ultimate prognosis depends much more upon the primary cause of the pleurisy than upon the extent of the pleural inflammation. Cases with a severe and sudden onset often recover more completely and rapidly than the insidious types. This is only another way of stating that the pleurisy caused by the bacillus of tubercle is at the best of slow duration, and in the long run usually proves fatal, though recovery is by no means rare in true tubercular pleurisy, as in tubercular peritonitis.

The examination of the exudate affords therefore the best evidence upon which to base an accurate prognosis. Pneumococcic pleurisy, even when ending in empyema, should be much more favourably regarded than the streptococcic. In children, a hopeful prognosis may confidently be given when the pneumococcus alone is present, many cases, even when purulent, recovering after a single tapping. The danger in the streptococcic cases is of septicæmia or pyæmia supervening after the exudation has become purulent; the prognosis therefore becomes serious when hectic symptoms and rigors appear during the third week, especially after the sac has

been opened. A hæmorrhagic exudate is generally an evil omen. A sterile exudate should always be regarded as serious for the reason already stated—viz. the great majority of such cases are tubercular.

Sudden death is an occasional event even when the effusion is serous and not excessive, being due to a large thrombus forming in the right auricle or ventricle. In rare cases sudden death has followed some kinking of the great vessels close to the heart. It has sometimes been reported as following soon after tapping, but this result is about as rare as is the fatal suffocation following a rupture of the sac into a large bronchus.

The prognosis of an ultimate complete recovery is very doubtful where the effusion has been permitted to remain for many weeks, causing complete carnification of the lung. Such cases are especially prone to eventually terminate in phthisis—a final event never to be lost sight of in all cases of pleurisy from whatever cause arising. Double acute pleurisy, except that which arises occasionally during an attack of rheumatic fever, justifies a very grave prognosis.

Treatment.—The indication in the first stage of the disease is the relief of pain. Morphia hypodermically is the most effective agent, and it should not be withheld when the suffering is severe. Leeching, cupping, iced compresses, hot poultices, blistering, and strapping of the affected side all have their advocates, and one measure may be resorted to when another fails; thus when a hot poultice does not afford relief, ice may be tried.

General blood-letting in the severe pleurisy which sometimes follows broken ribs has given in the hands of the writer most striking relief. Measures to combat the severity of the fever may be indicated, and in smart cases aconite in small and frequently repeated doses is most beneficial; and a good routine treatment is a combination of this drug with salicylates. The general hygienic and dietetic management of the case is to be carried out upon the lines indicated in any mild fever.

When effusion has taken place little reliance need be placed in drugs; but with a very moderate amount of exudation, which does not show signs of increasing, one large or a series of small flying blisters may be tried. These relieve pain and often seem to hasten absorption. Iodine in mild and chronic cases may be applied to the chest-wall, and iodides internally given at the same time. Diuretics, as digitalis, diaphoretics, saline purgatives, and dry dieting have seldom any effect upon an inflammatory exudation, though they may rapidly cause the absorption of passive exudations in ordinary hydrothorax.

There is a consensus of opinion that, in all cases of extensive effusion, early, but not too early, resort to aspiration

should be the rule. More frequently the error is made in delaying too long than in operating prematurely.

The fluid should be removed in all cases where one pleural sac is found to be full when the patient first comes under observation. With a dulness extending above the level of the angle of the scapula which has lasted for three or four weeks, the indications are equally clear. Marked displacement of organs, even when the signs of fluid do not appear to indicate that the sac is greatly distended, demands aspiration without delay, and the operation should be performed in double pleurisy when each sac is only half full.

The danger of sudden death in such cases, if left alone, though remote, should prevent a policy of procrastination; and, moreover, there is always the liability of the compressed lung becoming bound down permanently by adhesions. It is a good rule to only remove about half of the exudation, and to do so as slowly as possible, only using as little aspirating force as is necessary to maintain a steady flow from the cannula. If the method of syphonage by means of a fine Southey's tube be employed, the entire evacuation of the fluid may usually be safely effected.

The best site for the puncture is in the lateral region of the thorax in the mid-axillary line in any intercostal space from the fourth to the eighth. The mistake is likely to be made of puncturing too low down. The patient should be in the lying posture, with the head slightly raised and the affected side of the thorax uppermost, with the hand resting upon the opposite shoulder. In all cases it is advisable, when using a trocar and cannula, first to locate the fluid by puncture with the needle of an ordinary hypodermic syringe. When this precaution is exercised the aspiration may be made at almost any spot in the chest-wall. It is advisable to penetrate the tissues close to the upper border of the rib or fair in the centre of the intercostal space, so as to avoid the intercostal artery.

Should coughing occur, the flow should be stopped for a short time without withdrawing the cannula; but excessive coughing, dyspnoea, or pulmonary embarrassment is an indication that some hyperæmia or œdema of the lung is taking place, and the operator should withdraw the tube and trust to nature for the removal of the residual fluid, or repeat the operation after the lapse of several days. The operation is seldom followed by any untoward event, but very occasionally air may be admitted to the pleural sac, which, however, is followed by no evil consequences, as a rule. Sometimes air may find its way into the subcutaneous tissue, causing an emphysematous condition around the site of puncture. Pneumothorax very seldom occurs from perforation of the lung-tissue, but a frothy expectoration may be brought up in abundance towards the end of the tapping, and should be

an indication for the immediate cessation of the aspiration. When this follows the operation it constitutes the so-called 'albuminous expectoration,' which is the result of an acute oedema of the lung, and which has been known to terminate fatally. Syncope is a not altogether rare occurrence, but it probably never occurs save when the fluid has been aspirated too quickly or under too great pressure.

A second tapping may be necessary, and in rare cases a succession of these may be demanded ; but many authorities recommend in such chronic cases that a free incision should be made and drainage established, though the fluid be fibrinous throughout.

The treatment of empyema is described under its own heading. As a rule, a free incision with drainage is imperative as soon as the aspirating needle has revealed the presence of pus ; though in the case of the pneumococcic infections of children a suppurative effusion should have the chance of at least one removal of the purulent exudate before resorting to incision.

The after-treatment of pleurisy is a subject the importance of which can hardly be exaggerated, owing to the frequency with which phthisis supervenes. Every means found beneficial in the tubercular condition should be rigidly adhered to, as improved hygiene, gymnastics, free open-air life, good living, &c. The earliest full expansion of the compressed lung and of the retracted side of the thorax should be encouraged by resort to pulmonary gymnastic or thorough breathing exercises.

PLEURODYNIA. (See under RHEUMATISM, CHRONIC.)

PLUMBISM. (See under LEAD-POISONING.)

PNEUMONIA, ACUTE

As implied in its old synonym of *pneumonitis*, this is a form of fever characterised by an acute inflammation of the lung, now recognised as the result of specific infection, either single or multiple.

In a wider sense, all inflammatory affections of the lung are still grouped together under the name of Pneumonia, whether these be acute or chronic, primary or secondary, lobar or lobular, parenchymatous or interstitial. A classification upon a strictly anatomical, etiological, bacteriological, or clinical basis is still surrounded with considerable difficulty, since the same cause may produce different types of the

disease. For all practical purposes, however, the description of the acute lobar type of the disease constitutes a clearly defined clinical picture for the most part easily recognised at the bedside and included in the above definition. The chronic forms will be described afterwards.

ACUTE LOBAR or GROUPOUS PNEUMONIA, or PNEUMONIC FEVER

Etiology.—The trend of opinion is certainly in the direction of regarding the etiological factor in *all* cases of typical croupous pneumonia to be the presence of the *diplococcus lanceolatus* of Fraenkel, also known as the pneumococcus. Bovaird has found this organism in 95 per cent. of the cases investigated by him. The other microbes sometimes present will be referred to in the description of the bacteriology of the affection, and the question of the essential unity of the disease will be there discussed.

The disease is one of the most widespread, occurring in all climes and at all seasons from the earliest times. By many authorities its incidence is considered to be increasing. Ziemssen states that it may be accepted that acute pneumonia constitutes 3 per cent. of the total of all diseases met with in hospitals; and the same figure is believed by Aufrecht to hold true of the total of all human diseases. The increase may be partly accounted for by the fact that the prevalence of the disease is decidedly greater in the denizens of crowded cities, amongst whom the mortality is always higher than in the rural districts. Of late years the prevalence of influenza seems to have influenced the frequency and increased the death-rate of pneumonia.

Predisposing Causes.—*Season* shows an unmistakable influence, the number of cases being greatest in temperate climes in the months of March, April, and May. *Cold* is an etiological factor obvious in the history of many cases in which the attack has rapidly followed severe exposure or a thorough wetting in cold weather. With the consideration of this cause may be associated that of season, as it is noteworthy that the disease is more prevalent in the spring months when the changes of temperature are more violent and the percentage of moisture in the air more fluctuating than in the midwinter months. These changes act like direct exposure of the body to cold and wet, and are accepted as operative by their power of reducing the normal vital resistance of the individual. By some authorities, as Netter, the increased incidence of the disease is explained by the theory that the pneumococcus is increased in virulence during the spring months. Moore points out the important fact that the prevalence or mortality of pneumonia is in marked contrast with that of bronchitis, which reaches its greatest height in mid-winter and fades with the advent of spring.

Age.—No age is exempt from the disease. Sir John Moore's Irish statistics for five years prove that up to the fifth or sixth year the number of deaths registered from acute pneumonia constitute nearly one-fourth part of the total pneumonic death-rate, the majority of the deaths in the first quinquennium being those of infants of one year and under. Between the ages of ten and fifteen the rate diminishes, after which period it steadily increases, when the proportion of patients living to an advanced age is considered. The difficulty in arriving at any accurate conclusion from statistics is seen in a contrast of the above figures with those of Pye-Smith, derived from hospital-cases; he sums up his results by stating: 'Lobar pneumonia is rare in infancy, very common between two years and twenty, gradually less common in adult life, more rare after fifty.' It is, however, possible that of the deaths in children of one year and under in the above returns of the Registrar-General for Ireland many were not really due to pneumonia of the true lobar or croupous type.

Sex.—Males are more frequently affected than females, probably owing to their greater opportunities for exposure. In childhood both sexes suffer about equally; after puberty the rate of incidence amongst males steadily increases.

Habits.—City-life predisposes to the disease, and it occurs with greater frequency amongst the hard-working outdoor operatives and labourers, especially amongst those addicted to alcoholism.

Other Diseases.—Influenza, typhus, and typhoid fevers, diabetes and Bright's disease, must be regarded as etiological factors or predisposing causes, since acute croupous pneumonia is more liable to attack patients suffering from these affections. Measles and pertussis, upon the other hand, are more frequently complicated by the pneumonia of the lobar type. *W. J. J.*

Trauma.—In a small percentage of cases the disease has followed some severe injury to the chest-wall, as a violent blow or contusion without breach of skin or apparently without any direct injury to the lung.

Contagion.—Though examples of the direct spread of the disease under ordinary conditions from a pneumonic patient to healthy individuals coming into close personal contact with him are almost unknown, nevertheless the infectious nature of some varieties of pneumonia is beyond doubt. This is proved by the occasional spread of croupous pneumonia in barracks and gaols and its appearance in epidemics, where its infectious nature can be often traced by its transmission from one person to another. It would appear, however, that some factor is at work in these cases which is absent in the common type of endemic croupous pneumonia, since the highly infectious character of the disease in certain epidemics

is a very obvious feature. Early in the recent pandemic of influenza the writer drew attention to the highly infectious nature of the acute lobar pneumonia which often complicated the disease, many healthy individuals coming into contact with the patient being seized with the complication, whilst apparently escaping the primary malady. This infectious character seen in some epidemics of apparently typical acute croupous pneumonia has led some authorities to still believe in the pleurality of the infection.

Influence of Previous Attacks.—It appears probable that one attack of the disease renders the patient more liable than if he had never suffered, but it is possible that the numerous recurrences might be explained by some hereditary or individual predisposition to the disease. Cases are on record where the same individual has experienced as many as half a score of distinct attacks. It is calculated that probably one-fourth of the patients suffering from pneumonia have experienced former attacks.

Symptoms.—The incubation-period of pneumonia is apparently a very short one. In the cases where direct contagion has been traced the period has been found to vary from a few hours to not more than three days. In some the invasion has been observed to follow almost immediately upon the supposed exposure. The specific pneumococcus loses its virulence within a week in laboratory-cultures, and Sir John Moore therefore points out that it is almost certain that the period of incubation cannot extend beyond five or six days.

In this period there are often no symptoms, the invasion setting in with marked abruptness; but occasionally a brief period of malaise, sometimes associated with slight catarrhal discomfort, may be experienced by the patient before the initial rigor, which is usually severe and sometimes as violent in its intensity as the rigor of malarial fever. In children the place of the rigor is usually taken by a severe general convulsion. Headache, muscular pains, thirst, a hot burning skin, and furred tongue, with a feeling of great prostration or profound depression, often associated with vomiting, soon follow, so that within an hour after the patient has been in the enjoyment of perfect health he may be obviously very ill. Pain in the side is a very early symptom; sometimes it is experienced at the onset of the rigor. It is lancinating and severe, like the pain of acute pleurisy, and greatly intensified by deep inspiration. The breathing becomes shallow and quick, and a short dry cough develops which is often very distressing owing to its effect upon the pain. This latter is usually referred to somewhere about the nipple-region, but it may be felt in the abdomen or even, in rare cases, on the sound side of the thorax, and is probably the result of con-

comitant pleurisy. Fever is present from the first moment of the onset; during the rigor, or a few hours after, the temperature may reach 102° or more, and it continues to steadily rise till a high degree is registered.

By the expiration of twenty-four hours from the initial rigor the clinical picture in severe cases is usually well defined in its main outlines. The patient lies flat upon his back with his shoulders slightly raised, if he has not already discovered, should his apex be involved, that lying upon his affected side gives him some slight ease in breathing; but dorsal decubitus is the rule in pneumonia of the base. His eyes are lustrous, his face is flushed by a crimson blush over each malar prominence, his expression is anxious, and the respirations quick, shallow, and obviously painful; herpes is commonly seen upon his lips, the vesicles developing rapidly upon a hyperæmic base as in the herpes of ordinary severe catarrhs. The malar blush is not infrequently confined to the side affected with the pneumonia. The pulse is full and of increased tension; it is accelerated at this stage, but not in proportion to the intensity of the fever or to the frequency of the respirations. This latter is an important characteristic phenomenon in pneumonia, the respirations often being as many as 50 in the minute, when the pulse-rate may not exceed 100: the ratio being thus one to two instead of the normal one to four. The skin is hot and feels burning to the hand of the examiner, as it only does in the dry stage of malarial and scarlatinal fevers, there being complete absence of moisture with the increased production of heat. The temperature by this time has generally reached a height of 104° or 105° , and it remains often at about 104° throughout, with but a slight fluctuation of half or one degree. The type of pneumonic fever is therefore very markedly *continuous* and high-sustained. In adynamic cases the range of fever is sometimes of a milder type, which is, however, not a favourable sign; and often in the aged a fatal pneumonia may run its course without any pyrexia whatever.

Cough is now well marked, but it is no longer dry. A little mucus tinged or streaked with bright blood is brought up usually within the first twelve hours; sometimes the attack commences with a smart hæmoptysis. This is soon replaced by the typical sputum of pneumonia, which presents the following characters: It is viscid, tough, tenacious, and gluey, adhering to the patient's lips and removed even with difficulty from the polished surface of the spittoon. Brick-red or rusty in colour from the intimate mixing up of the blood-cells with the mucus and epithelial constituents, it can scarcely be confused with the sputum met with in any other condition. In the great majority of cases it is present, generally appearing before the termination of the first twenty-four hours after

the initial rigor. The total quantity expectorated is small, very seldom reaching five ounces in the day and night; more frequently somewhat less than half this amount is brought up. Sometimes it is greenish-yellow, constituting the so-called 'greengage' expectoration, which is often associated with some degree of jaundice.

A valuable aid to prognosis may be obtained by careful examination of the physical characters of the sputum. In adynamic cases, especially in alcoholic subjects, it is more fluid and of a dark greenish-brown colour, resembling prune-juice; this is often a serious, though not necessarily a fatal, sign, as is also an almost similar condition of the sputum resembling a tobacco-spit. In children often there is no expectoration, since the sputum is usually swallowed; occasionally only in adults is it entirely absent, and this occurs most frequently in apical pneumonia. In some cases considerable plugs of fibrin are visible in the expectoration; and after the appearance of these, should the sputum become scanty or disappear whilst coughing increases, the probability is that the bronchioles have become plugged. A considerable amount of free blood is, however, not an unfavourable sign. Recently the writer has seen death result directly from a large hæmoptysis in an elderly subject upon the eighth or ninth day of the disease.

Under the microscope are found red corpuscles, mucus-cells, leucocytes, and epithelial elements, all in various stages of degeneration and held together by a meshwork of fine fibrinous exudation. Pneumococci, as a rule, are present, usually in association with other organisms. After the crisis the expectoration loses its rusty-red colour, becomes less tough and more abundant, gradually acquiring the greenish-yellow colour of bronchial expectoration and the nummular character of phthisical sputum, sinking in water owing to the absence of air. As resolution advances it becomes less coloured, and finally purely mucoid before passing away.

The typical clinical picture above described maintains its chief features unaltered, though mild delirium may occur, the temperature remaining continuously high with but slight remissions till some time about the seventh day, when the *crisis* is reached; the fever and other symptoms subside as rapidly as they appeared. From the earliest times the crisis has been believed to occur much more frequently upon the odd days than upon the even ones, and modern statistics prove, in the main, the correctness of this early statement. Ziemssen found that the crisis began upon an odd day in 95 out of 107 cases; other observers, however, report a much larger minority upon the even days. The duration of the fever varies from three days in rare cases to a crisis occurring upon the fifth, seventh, ninth, or eleventh day. The drop

in the temperature commonly sets in late in the evening, and the fever is often found to have entirely disappeared by the following morning, when a sub-normal figure is reached. Sometimes the crisis is even more abrupt and sudden, the drop from 104° or 105° to 97° occurring within a couple of hours. Occasionally the recovery is by lysis, there being a slow and steady fall, spread evenly over twenty-four hours or longer—a method of termination, upon the whole, more likely to occur in cases protracted beyond the ninth day, especially when some complication is present. The crisis is accompanied usually by profuse perspirations, after which the patient often falls into a peaceful refreshing sleep, and awakes with a feeling of being free from his trouble, though the physical signs in the lung may show little change for a few days afterwards. The pulse may fall to 60 and the respirations to 20.

It is not rare for a sharp rise in the fever immediately to precede the crisis, and occasionally a fall occurs (pseudo-crisis) before the defervescence; this fall is followed by a marked elevation, which may last for twenty-four or thirty-six hours before the real crisis sets in. In children and aged subjects the course is usually less typical than that just described, and it is not an infrequent experience to find that cough, pain, and sputum are all absent.

Physical Signs.—*Inspection* will reveal some diminution of the chest-movements upon the affected side, but as a rule no change in the intercostal spaces will be visible, and, though the side may appear to the eye to be enlarged, it only measures a very little more than its sound fellow. The signs upon inspection in the early stage will be mainly those caused by the accompanying pleuritis, but the deficient movement will be much less marked than in pleurisy with effusion, though the dyspnœa or frequency of the respirations is usually more obvious. The decubitus is upon the back, or the patient lies upon his affected side. The degree of dyspnœa may be gauged by the play of the nostrils, which dilate in inspiration.

Palpation.—This will corroborate the diminution of the respiratory movements visible upon inspection, and sometimes the observer may satisfy himself that the movements of the thoracic wall are normal over the upper part whilst markedly diminished over the base of the affected side of the thorax. The fingers should appreciate a distinct difference in palpating the intercostal spaces, the resistance being always greater upon the affected side, though this is more obvious as a rule upon percussion. This increased resistance is less than in pleural effusion. Some observers maintain that by the fingers an increase in the temperature may be appreciated over the inflamed lung, and that it is possible to map out the limits of the pneumonic area by this method of

thermo-palpation—a feat which the writer has never been able to accomplish.

Rarely is a friction-fremitus to be detected, but, as a rule, the voice-fremitus is markedly increased—a condition of great value in the exclusion of fluid effusion. Even when absent, owing to plugging of the bronchi by fibrinous masses, it returns speedily after a severe cough. Sometimes the cardiac pulsations can be distinctly felt over the base of the lung, being transmitted through the consolidated tissue.

Percussion.—In the preliminary stage and in the stage of actual engorgement, before consolidation has set in, the side on percussion is somewhat hyper-resonant, or even tympanitic. This phenomenon is explained by the diminution in the normal elasticity of the lung-tissue, as the organ is in a relaxed condition owing to the distension of its capillaries with blood. When the stage of red hepatisation is reached the note is distinctly dull, unless in central pneumonias, where a deep stroke is required to bring out any dulness. The dull sound over a pneumonic lung has never the dead flatness elicited by percussing a pleural effusion, nor is the sense of resistance of the tissues as conveyed to the finger ever so great. Indeed, most authors agree that in the dull sound produced by percussing over a consolidated lung a hollow, tubular, or tympanitic quality may always be detected in it; this is usually evident in apical pneumonias. Over the apex of a lung whose base is solidified occasionally a cracked-pot sound is elicited, and there is often marked tympanitis amounting to Skodaic resonance; sometimes a similar hyper-resonant note may be heard on striking the sound side. There is markedly increased myotatic irritability from the earliest stage of the disease, as elicited by percussion of the fibres of the pectoralis major on the affected side, as first pointed out by Kennan and corroborated by Sir John Moore.

Auscultation.—In the earliest (the preliminary) stage the breath-sounds may be diminished or increased—a matter of little clinical signification, since it only lasts for a few hours. In one case the writer chanced to examine a patient immediately upon the onset of the disease, and he found the puerile breathing described by Stokes, over the base of the lung which afterwards became solidified. The presence of diminution or intensification of the breath-sounds appears to the writer to depend upon the degree of pleurisy present at the very beginning of the disease.

The most important diagnostic sign is the presence of the *fine crepitant râle of Laennec*. The sound is made up of a series of fine crackles or minute explosions, and can be identically reproduced by rubbing between the finger and thumb a lock of hair above the observer's ear. It conveys the idea of nearness, being apparently produced in the pneumonic lung

close to its surface—a quality which has induced many authorities, including Osler, to regard it as a fine pleural crepitus. It is, however, almost certainly caused by minute bubbles of air in the viscid secretion contained in the minute bronchioles or by the separation of the walls of these or of the air-cells during inspiration after they have been covered over with the glutinous exudation characteristic of the early stages of the disease. The sound is only heard towards the end of inspiration, and is usually present as soon as the patient comes under observation. Though it disappears upon consolidation, it may nearly always be heard about the margin of the consolidated area for a considerable time after it has been replaced by other sounds over the affected lobe.

This fine crepitant râle, though highly characteristic of acute croupous pneumonia, cannot be considered as absolutely pathognomonic, since an identical sound is heard in pulmonary oedema, hæmorrhagic infarct, hypostatic engorgement, capillary bronchitis, and sometimes in phthisis. But when its localisation or distribution is taken into consideration along with the constitutional symptoms, it may with safety be taken as conclusive evidence of croupous pneumonia.

After the advent of consolidation (red hepatisation), the prolongation of the expiratory murmur heralds the advent of *bronchial breathing*, which becomes well marked in the majority of cases by the end of the third or the beginning of the fourth day. The vesicular murmur is entirely replaced by the high-pitched, dry, blowing, harsh sounds heard in inspiration and expiration, the latter being generally the higher in pitch, and heard as if breathed into the ear of the auscultator. The sounds are of about equal length and loudness and are separated by a short but distinct interval. The phenomenon thus somewhat closely resembles that observed on listening over a large bronchus, and when present in its most typical state or degree of greatest intensity it is usually styled tubular breathing, and more closely simulates the sounds heard over the normal trachea. With these it is practically identical, being the result of the sounds produced in the larynx and communicated with great distinctness through the columns of motionless air in the bronchi, which are surrounded by the excellent conducting medium of the pneumonic consolidation.

The sign is absent when the bronchi are filled with adhesive exudate or fibrinous plugs, which prevent the transmission of the breath-sounds by the air; but it may return after voluntary expulsive efforts. It is not rarely entirely absent in apical pneumonia, in which condition the solidification of the lung is often incomplete.

Bronchophony, usually of a high-pitched character, is present, the voice-sounds of the patient being conducted to the ear with marked intensity. To this intensification of the voice-

sound is often added a peculiar wavering bleating character or nasal twang, known as *ægophony* or bleating bronchophony. Aufrecht regards *ægophony* as an expiratory staccato interruption of the bronchophony over the consolidated lung analogous to the healthy speech of those who possess a tremulous voice. Though the phenomenon is often met with in pleural effusion, the presence of fluid in the pleural sac is certainly not necessary for its production. *Pectoriloquy* is also present. This is the term applied to the condition in which not only the laryngeal voice-sounds but the articulated word-sounds produced in the mouth are audible. Thus if the patient whispers, the mouth-sounds alone are audible, in which case the phenomenon is sometimes described as *whispering* pectoriloquy. Tussive resonance is also greatly increased.

The heart-sounds are audible with great distinctness, especially in left-sided pneumonia, owing to the increased conducting power of the hepatised lung.

The bronchial breathing and bronchophony are usually unaccompanied by râles or rhonchi, unless there happens to be an unusual degree of catarrh of the bronchial surface. The friction-sound of the accompanying pleurisy is often to be detected.

As the stage of resolution sets in with the passing off of grey hepatisation, the characteristic *redux crepitation* is heard. This is a consonating or sub-crepitant mucous râle, coarser than the fine crepitant râle of the first stage usually, but sometimes it is as fine as the latter; it, however, may be heard in both inspiration and expiration, whilst the fine pneumonic crepitation is confined to inspiration.

As resolution proceeds other coarser râles are audible upon the further opening-up of the air-vesicles and small bronchi. Very rarely are adventitious sounds audible over the sound side. The signs of dulness, bronchial breathing, &c., may remain for several days with little change after the total cessation of fever and constitutional symptoms, but they always pass off, leaving the lung in a normal condition. Occasionally pleural effusion, an abscess in the lung-substance, or a localised empyema may complicate the physical signs in the later stages of the disease and retard the advent of the crisis. Connor and Dodge found that delayed resolution, as manifested by the signs remaining for two weeks or more, occurred in 15 per cent. of their cases.

Blood-examination.—Leucocytosis is well marked in the disease; as a rule, the number of white blood-cells is found increased from the beginning, and usually in proportion to the height of the fever. The count may even reach 50,000 in rare cases; it touches its highest figure just before the crisis, and unless some suppurative complication be present there

is a sudden drop in the number, which then steadily falls to the normal. The point is of some value in the prognosis, as in the malignant type of the disease leucocytosis is absent throughout.

The pneumococcus is found in the blood, according to some observers, in *every* case when a considerable amount of blood is used in the investigation.

Agglutination or serum-reaction, as in Widal's typhoid-fever test, has been employed by some; but the results are of little clinical value, since the reaction is not reliable unless the pneumococcus be obtained in the first instance from the blood or sputum of the individual whose serum is to be afterwards tested.

Radioscopy.—The X-rays are valuable in the diagnosis of doubtful cases, the affected portion of the lung showing considerable darkening and revealing the diminution in the diaphragmatic movements.

The above description of the symptoms and signs applies to a typical case of the disease ending in recovery after a crisis occurring about the end of a week. The pulmonary signs afford evidence of the extent of the local lesion, but the constitutional symptoms—fever, pulse-rate, delirium, &c.—are the measure of the degree of the general toxæmia, and these may not be proportionate to the local signs. The toxæmia is obviously the result of the poisonous products of the pneumococcus, which interfere with the functions of the different organs. The variation in the symptoms in the different stages of the disease up to the period of the crisis may be regarded from the point of view of the local lesion as of little clinical signification, it being the manifestation of the general toxæmia; hence, at the bedside, the anatomical or pathological division of the stages of pneumonia into those of engorgement, red and grey hepatisation, and purulent resolution is of little assistance.

These constitutional symptoms may be referred to in detail.

Fever.—Little further need be added to what has been already stated upon page 1399. The characteristic of the fever is its continuous type, with slight morning remissions; but often a marked remission occurs upon the second or third day, when the temperature may fall to the normal and remain at this figure for twenty-four hours, after which it rises rapidly to almost its former height, and remains, with slight remissions, to the crisis.

Circulatory Symptoms.—These are of greatest importance, and a study of the cardiac condition affords the best means of judging of the probable outcome of the attack. The pulse, as already stated, is accelerated, but not in proportion to the frequency of the respirations. At a later stage its marked

acceleration may usually be regarded as a very serious sign.

In children from the onset it becomes very rapid, and in them especially the mere frequency of the pulse affords no criterion of the severity of the disease. In adults, in grave cases, the cardiac pulsations may even number the same as the respirations.

Of more importance is the *character* of the pulse, though little significance need be attached to marked dirotism, which is sometimes observed; but irregularity setting in about the fifth day before the crisis cannot but be regarded as an unfavourable omen. A similar irregularity occurring after the cessation of fever is of little consequence.

The effect of the toxins upon the heart-muscle or upon its nerve-supply or upon the vaso-motor centres may induce serious changes in the circulation; and it must be accepted as true that sudden collapse occurring at any stage of the attack is very much more frequently the result of cardiac failure than of the effects of the toxins upon the brain or other organs.

Rapid degeneration of the myocardium occurs in all severe toxic cases; this leads to dilatation of the heart on the right side caused by the pulmonary embarrassment, whilst the left ventricle, owing to the diminished amount of blood which it receives, is less liable to distension until a general blocking occurs or till from some unusual toxic influence the blood-pressure rises, when the already poisoned cardiac muscle staggers under its increased load; but the blood-pressure is usually reduced very considerably when the symptoms of severe toxæmia are present.

The cardiac weakness may show itself by the galloping rhythm described by Fräntzel, in which three sounds instead of two are heard over the weakened organ.

Sudden cardiac failure may occur at any stage; it may be seen in the change of the face, which becomes anxious, and ashy-grey in colour; the tip of the nose, ears, hands, and feet get cold, though the temperature in the axilla or rectum may be very high. The characters of the pulse at this time should be closely noted; it is invariably much accelerated, very small and thready, and often irregular—changes which call for free stimulation. At the crisis also, or very soon after, collapse from cardiac degeneration or muscular failure is not a rare event. The symptoms then are different, inasmuch as the collapse sets in with a sub-normal temperature, profuse sweating, dyspnœa, cyanosis, and sometimes with a drop of the pulse-rate to less than half its former frequency. Recovery may take place after these sudden collapses when prompt action is taken, but the steadily progressive cardiac asthenia which shows itself by a gradually increasing dilatation of the

chambers of the heart and great pulmonary embarrassment always proves fatal.

Nervous Symptoms.—These vary considerably, and, like the cerebral disturbances met with in other fevers, appear to be largely influenced by the individual's mental traits. As a rule, delirium is not marked except at night or for a short time after awaking from sleep in the daytime. In alcoholic patients it takes on the characters of delirium tremens, and, with the other asthenic symptoms, is a most unfavourable omen. Headache, restlessness, and insomnia are present, however, in all cases, and the headache is greatly intensified by coughing. In children, the initial convulsion may be succeeded by delirium, cerebral vomiting, stupor, stiffness of the neck, and other symptoms of meningeal mischief, though the after-progress of the case demonstrates that inflammation of the meninges was absent throughout. Both in children and adults hallucinations are liable to be present, so that the pneumonic patient should always be most closely watched.

Apical pneumonia is unquestionably more liable to be associated with a severe delirium, which often takes on the character of acute mania. The intensity of the delirium and other nervous symptoms often bears no relation to the high degree of the pyrexia. This fact is demonstrated by the appearance of wild delirium supervening sometimes after the crisis has satisfactorily terminated. In a very small proportion of these cases a temporary insanity may follow even after all the lung-signs have cleared up. When renal disturbances are marked, uræmic symptoms may be added on to the delirium; but uræmic convulsions and coma are rare, except in the pneumonia which sometimes occurs as a terminal event in Bright's disease and diabetes.

Pneumonic hemiplegia occurs in a small percentage of cases; it is found more frequently in apical pneumonias, and has been met with in children. It can be differentiated from the apoplectic seizures which result from embolism, hæmorrhage, or thrombosis, since it passes off speedily as the circulatory disturbances improve.

Cutaneous Signs.—The appearance of herpes has been already referred to; it occurs in nearly half the cases, and the site is most commonly the junction of the skin and mucous membrane on the nose and lips; but it is not rare on the ears and chin, and has been observed on the anal margin and prepuce. It may precede the attack or follow the crisis, but most usually the herpetic eruption is out upon the third day, and by many is regarded as a favourable sign.

Perspiration may be very copious and associated with sudamina; as a rule, it is absent in the early stages, but the crisis is usually accompanied by drenching sweating. A slight

degree of icteroid tinge is common in the skin and conjunctiva, and sometimes cyanosis may be marked.

Urinary Changes.—These correspond, in the main, to those observed in other fevers; the quantity is diminished, it is of high colour and s.g., and markedly acid. It becomes more copious after the crisis, unless the sweating is excessive, and then the urea is much increased, the urates being deposited heavily, and, if the urine be highly acid, there is usually a considerable amount of uric acid also deposited.

Chlorides are much diminished throughout the attack, and may be entirely absent from the urine, but appear after the crisis in considerable amount—a fact of some diagnostic importance. Albuminuria is often observed, usually of slight degree and fluctuating in proportion to the severity of the fever; casts may also be detected, but unless occurring in abundance and associated with blood and much renal epithelial debris, they are not to be regarded as evidence of nephritis. Peptonuria is often present after the crisis, and the diazo-reaction may be sometimes well marked.

Digestive Disturbances.—Vomiting, especially in children, is a common symptom; but it is rarely seen except in the very early stage, unless some complication, as meningitis, is present. The tongue is coated with a yellowish-white fur, and as the fever becomes high it tends to get brown and dry. Thirst is marked, appetite diminishes or is entirely lost, and constipation is the rule, but the crisis may be accompanied by a severe attack of diarrhoea. Abdominal pain is sometimes marked, and is usually associated with a considerable degree of tympanitic distension, so that the lung-symptoms may be overshadowed, the clinical picture simulating that of a severe appendicular inflammation. This is always a grave sign owing to the already embarrassed condition of the respiration. The abdominal pain is explained by Udalpho on the theory of an anatomical connection between the sympathetic or intercostal nerve-supply of the appendix-region and that of the involved pulmonary area. The liver is depressed and sometimes appears to be enlarged, and the spleen can often be detected upon palpation.

Morbid Anatomy.—The predominant changes observed in the lung in croupous pneumonia depend upon the stage of the disease at the time of death. Pathologists now recognise the four well-marked stages, following, in the main, the pathological classification of Laennec. These are: (1) The stage of inflammatory engorgement, (2) red hepatisation, (3) grey hepatisation, (4) the stage of resolution, sometimes named that of purulent infiltration, Laennec's and Rokitsansky's original 'hepatisation' stage being now differentiated into the red and grey stages. All these processes may, however, often be found in the lung at the same time, one stage being

visible to the naked eye in a particular area, in its transition into the next process which may be found in its typical state close by.

1. The typical condition of *inflammatory engorgement* is seldom met with, since death during the first or second day of the disease is an extremely rare event, nevertheless the frequency with which the change is observed in the spreading margins of the solidified pneumonic lung leave no doubt about its being invariably present at the beginning of the disease.

To the naked eye the lung is filled with blood; it is of dark red or purple colour and is of increased consistence; it pits on pressure, is inflatable, does not collapse on removal from the body but retains its shape, and tears readily, though it eripitates, and does not sink in water. Upon incision, dark-coloured blood and serum ooze from the cut surface, which becomes a brighter red after exposure to the air. The microscopic appearances correspond with the macroscopic. The capillaries are distended with red cells, and the alveoli are filled with desquamated alveolar epithelium and a small quantity of red blood-discs. From the purely pathological standpoint much interest attaches itself to the nature of the process in this first stage of the disease.

The commonly accepted view, that the stage of engorgement is one primarily of hyperæmia of the pulmonary capillaries, is challenged by Aufrecht and by Heitler. The former authority produces histological evidence, which seems indisputable, that the nature of the pathological process must be regarded as an inflammation of the alveolar epithelium. The first anatomical change effected by the disease, which belongs to the province of parenchymatous inflammation, is *followed*, and not preceded, by hyperæmia of the capillaries. The swelling and opacity of the alveolar epithelial cells and the increase in the number of their nuclei are soon followed by the outpouring of fibrin from the congested capillaries, which occurs at the commencement of the next stage.

2. The stage of *red hepatisation*. In this the affected lobe of the lung is solid and red, resembling somewhat the liver, as implied in its name. It is entirely devoid of air and is increased in bulk, the furrows or grooves upon its surface showing the impressions of the ribs. It sinks in water, and its weight is markedly increased, so that when the entire lung is pneumonic it may weigh as much as two to four pounds more than the healthy organ. Upon section it is firm, and presents a dark reddish-brown colour and a very distinct granular appearance. This latter is caused by the solid contents of the air-cells being squeezed out by the elastic tissue of the lung after section, the granules corresponding to the size of the alveoli. The cut surface is very friable and easily breaks down under the pressure of the finger.

The microscopic contents of the granulations, which measure about .2 mm., or of the distended alveoli, consist almost entirely of red blood-cells, with about the same sparse proportion of leucocytes as is found in normal blood. The cells are held together by a dense meshwork of fibrinous filaments, the complete formation being identical with a minute hæmorrhagic extravasation from the capillaries, whose walls have been weakened by the shedding of the alveolar epithelium, which has taken place during the stage of engorgement. The specific cocci may be detected amongst the cellular and fibrin elements of the alveoli.

3. The stage of *grey hepatisation* is recognised by its greyish-white or yellowish-grey colour; the cut surface of the lung is firm, friable, and granular. The granules separate more distinctly from the solidified tissue, being easily isolated with the gentlest pressure, and often not requiring any scraping or pressure to loosen or detach them from the alveolar cavities. Fibrinous plugs or bronchial casts are seen projecting from the cut mouths of the finer bronchi. When these are minutely examined they are found to be solid cylinders, and not tubes: this fact has led many to discard the term 'croupous' as applied to lobar pneumonia; but in the larger-sized bronchi true croupous hollow tubes are sometimes found, suggestive of the false membrane formation seen in diphtheritic conditions. The bronchi, when not plugged with fibrin, are usually found to contain a considerable quantity of thin, serous, frothy secretion.

The microscopic characters of the alveolar contents in the stage of grey hepatisation are in marked contrast with those observed in the red stage. They consist almost entirely of *white blood-cells*, the red corpuscles and the thick fibrinous network being only represented by a *débris*, which is suggestive of their former presence when this stage is fully established. Sometimes large phagocytic cells are present, the capillaries have lost their distended condition, and the alveolar wall is seen lined with its flat epithelial cells containing large nuclei.

4. The stage of *purulent infiltration* or of resolution is recognised by an advanced state of softening. Upon section no granules are visible, but a thin purulent secretion oozes from the alveoli, whose contents have become obviously liquefied and placed in a condition capable of being absorbed. That the softened exudate is removed in this way is rendered certain by the absence of any increase in the expectoration, and also by the increased elimination of nitrogenous products by the kidneys.

The changes observed in acute pleurisy are almost constantly found in the pleural membrane covering the affected portion of the lung; this has lost its polish, is thickened, and

usually covered with patches or with a continuous layer of fibrinous exudation. Only very occasionally there is seen a copious amount of fluid in the pleural sac. The bronchial glands are found enlarged and softened, especially in children. The morbid anatomy of the disease will often also reveal the changes in the pericardium and endocardium, whose inflammations frequently complicate the attack. The chambers of the heart are often found to contain firm white clots of fibrin, and obliteration of large branches of the pulmonary artery is not infrequently seen. The liver, spleen, and kidney usually show considerable hyperæmia and sometimes parenchymatous changes. The cerebral meninges and the peritoneum are occasionally found inflamed and sometimes the site of purulent secretions. Abscess of the lung is a not very rare result of the disease.

Pneumonia tends to involve the lower lobes much more frequently than the upper; it begins in the lower part of the lung and travels upwards, but the solidification is rarely strictly circumscribed by the fissures. The right organ is more frequently involved than the left. In nearly a third of the cases the hepatisation travels towards the apex, involving the entire lung. Sometimes the upper lobe is alone affected; in children it is not unusual for an apical pneumonia to spread downwards and involve the middle and lower lobe. Double apical pneumonia is rare, but both bases are often involved at the same time; and it is not very uncommon to find one base and the opposite apex to be the seat of solidification simultaneously. As already stated, it is a common event to find one lobe the seat of red hepatisation whilst another has already passed into the stage of grey solidification.

Bacteriology and Pathology.—The specific organism is found in almost every case. This is the Fraenkel-Weichselbaum diplococcus, also known as the *Diplococcus Pneumoniæ*, the *Pneumococcus*, or the *Micrococcus Lanceolatus*, which is not to be confounded with the organism described by Friedländer, and known as the *Bacillus Pneumoniæ*.

The diplococcus is an oval lancet-shaped organism, generally occurring in pairs, and provided with a distinct capsule. It is cultivated with considerable difficulty, apparently losing its vitality after a week or so in artificial culture *media*, but when certain precautions were taken the microbe was maintained alive and active by Emmerich and Foà for several weeks.

This diplococcus is found constantly, in almost pure cultures sometimes, in the diseased pulmonary tissue, in the blood, in the sputum, urine, fæces, and various regions of the body. It is accepted as the cause of croupous pneumonia, though it must be remembered that attempts to produce the disease in the lower animals by injecting the organisms have generally

ended in failure, though a septic condition always results. The diplococcus is frequently found in healthy individuals in the saliva and in the nasal and pharyngeal mucus; more than a quarter of a century has elapsed since Pasteur found it in the saliva. The upper respiratory tract may be accepted therefore as its normal habitation, and Polynère affirms that it exists in the healthy lung. What the causes are which render the innocent microbe virulent can be but conjectured, and a somewhat similar problem has been already discussed in connection with the usually harmless bacillus coli. The different agents, as season, cold, trauma, the incidence of other diseases, and the conditions mentioned under Etiology may be interpreted as predisposing causes which add to the virulence of the diplococcus or depress the natural resistance of the body. These agents, the writer suspects, may act by their using up or destroying some germicidal or antitoxic substance normally present in the patient's blood, and so permit the pneumococcus to rapidly multiply. This view is strengthened by a study of the development of the microbe, which is peculiarly sensitive to decay when cultivated in artificial media: its short-lived existence in the human body is obvious upon reflecting that pneumonia so often abruptly terminates as early as the fifth day and in some cases upon the third day. Another important fact may be also taken into consideration in this connection: it is certain that the pneumococcus may be the cause of acute pleurisy, endo- and pericarditis, meningitis, joint-inflammations, and peritonitis, in all of which conditions the lung-tissue may entirely escape. It is not improbable that the selection of the tissue in these latter cases is determined by the absence of the natural antitoxin in the fluids of the region involved.

The view of the *pleurality* of croupous pneumonia still insisted upon by Finkler, Weichselbaum, and a few others, and accepted as the result of carefully observed clinical phenomena by Sir John Moore, requires some notice. That other micro-organisms are frequently found in the lung, sputum and blood of pneumonic patients cannot be doubted. These are the pneumo-bacillus of Friedländer (which is also an encapsuled organism), streptococcus pyogenes, various staphylococci, and the organisms which produce diphtheria, typhoid fever, influenza, tuberculosis, anthrax, spotted fever, and plague. The modern view regarding the unity of the disease either accepts these latter-mentioned forms of pneumonia as *secondary* varieties of the disease, differing from the true croupous type, or they are accepted as cases of croupous pneumonia in which a multiple infection is present, the real causative agent—the pneumococcus—being invariably also found in evidence, as insisted upon by those who adhere to the pathological unity of the disease. The writer has

satisfied himself, especially in the case of the lobar pneumonia often seen in connection with influenza, that the physical signs and the highly contagious nature of the lung-affection *per se* are quite sufficient, in the majority of cases, to differentiate this special type of the disease from ordinary croupous pneumonia, whether the pneumococcus be present or not in conjunction with the influenzal bacillus (p. 712). Moreover, the unquestionably infectious nature of some epidemic forms of croupous pneumonia, if it does not justify a hesitation in accepting the dogmatic statement of Netter that croupous pneumonia can only be produced by the pneumococcus infection, should permit of the view being accepted that *clinically* more than one variety of so-called croupous pneumonia is capable of recognition at the bedside.

COMPLICATIONS.—These are either the result of the invasion of other organs or tissues by the pneumococcus or by the microbes which join with it in the dual or multiple infection.

Pleurisy.—As already seen, the presence of an early inflammation of the pleural membrane should be regarded as a part of the pneumonic condition, since its entire absence is probably an extremely rare event. It is, however, otherwise with the extensive serous or purulent exudations which sometimes make their appearance towards the termination of the attack; in this latter case the pleuritis may safely be regarded as a complication. The old term 'pleuro-pneumonia' is still employed to designate the condition in which from the onset the pleural symptoms and signs are so marked as to partially overshadow the pneumonic process. In the pneumonia of childhood the effusion is especially liable to end in empyema, but as a rule the cases yield to treatment after considerable prolongation of the period of convalescence. Pleurisy is sometimes seen as a typical complication of croupous pneumonia where the pleural inflammation attacks the side opposite to the pneumonia. In all cases the effusion produces the physical signs of pleurisy, but these may be masked by the dulness and other characteristics of the hepatised lung, so that the condition is liable to be overlooked. Delayed resolution or the presence of marked pyrexia appearing after the crisis should arouse suspicion of a complicating pleurisy. It must, however, be always borne in mind, as in primary pleurisy, that a large empyema may exist without fever; and this is especially true of the purulent exudation which not infrequently accompanies pneumonia in influenzal patients. Upon the other hand, a moderate serous effusion may cause a severe pyrexia of the continuous or remittent type.

Bronchitis.—A certain amount of bronchial irritation accompanies the pneumonic process, but rarely does it spread to the tubes in the unaffected lung, so that as a complication

it may be almost ignored; though in some epidemics a general bronchial affection has been reported, the explanation of which may be found in the assumption of a mixed infection in which the bacillus of ordinary catarrh played a prominent part.

Gangrene and Abscess of the Lung.—Pulmonary abscess occurs in but a very small proportion of cases; it arises from a breaking down of the lung-tissue in the area of grey hepatisation. The abscess-contents may be expectorated; more rarely, as in a case recently seen by the writer, the pus penetrates the pleura and causes a local empyema. The presence of yellow elastic fibres in the sputum is the only pathognomonic sign of this condition, which is liable to be mistaken for a perforative empyema. Gangrene of the lung is also a rare complication liable to be confused with a perforating empyema or a putrid bronchitis, and only to be differentiated from these conditions by the recognition of the *debris* of the lung-tissue found in the highly offensive-smelling expectoration.

Pericarditis occurs in a considerable percentage of cases; it was a much more frequent occurrence in the old venesection days, and Dietl has demonstrated recently that this method of treatment undoubtedly tends towards the production of pericardial inflammation.

The effusion is sometimes purulent, in which case a multiple infection may be suspected. The condition is liable to be overlooked, especially in left-sided pneumonia—a fact which explains its greater frequency in *post-mortem* records.

Endocarditis is a grave complication, fortunately of rare occurrence. Sometimes it takes on the malignant features observed in the ulcerative type of the disease, in which case the aortic valves are prone to suffer more frequently than the mitral curtains. Weichselbaum found the pneumococcus in the vegetations upon the diseased valves; probably sometimes streptococci or the micro-organisms causing acute rheumatism may be also present.

Jaundice.—This is often observed in acute pneumonia, and very different views are held regarding its cause. In some epidemics the icterus is a most marked feature, constituting the so-called 'bilious pneumonia.' The explanation of these cases is obviously that they are the product of a dual or multiple infection, and their mortality is very high. The question of whether this jaundice is of obstructive or hæmatogenous type need hardly be discussed, since the view is being generally accepted that all cases of toxæmic jaundice are the result of an obstructive process caused by the extreme viscosity of the bile. The jaundice which is seen in the typical croupous pneumonia occurring sporadically—that is, not in epidemic form—may be regarded as a more or less accidental

complication, the result of some catarrhal swelling of the common duct. It does not necessarily deepen the prognosis, unless there be evidence of previous liver-disease from prolonged alcoholic excesses. It certainly cannot be accepted as the result of a blood-condition produced by the absorption of the extensive exudation in the alveoli, as formerly held by some.

Abdominal meteorism, which is usually also associated with considerable pain, has been already referred to; it may or may not be the result of *peritonitis*, which is a comparatively rare complication. More frequently it appears to be caused by nerve-disturbances or reflected pain, such as is sometimes seen in the phenomenon of the pain being experienced in the sound side of the chest.

Diarrhœa is a rare complication, though occasionally present at the time of the crisis.

Meningitis is a serious complication; it is not rarely purulent and may be regarded as the direct result of the pneumococcus or some other associated micro-organism. It is occasionally the outcome of septic plugging caused by emboli in the malignant type of the endocardial complication. Weichselbaum's theory, that the pneumococcus reaches the brain membranes *via* the cellular tissue of the neck, is not necessarily the explanation of this complication, since the cocci are always found in the blood-stream, and staphylococci have been also found in the circulating fluid in cases where meningitis had complicated pneumonia.

Delirium tremens is not an uncommon complication in alcoholic patients who are attacked by pneumonia during or soon after the cessation of a prolonged debauch. It adds greatly to the gravity of the prognosis. It may in some instances be overlooked by the masking of the symptoms and signs owing to the predominance of the cerebral disturbance.

Peripheral neuritis has been met with as a sequela of the disease, and in a few instances local paralyses have been reported.

Thrombosis of the femoral or other veins occasionally occurs, and *gangrene* of a limb has been sometimes observed; possibly this latter rare complication may be the result of arterial plugging by the septic endocardial products, and a similar explanation may be applied to the *aphasia* and *otitis* which have been sometimes witnessed as complications.

Kidney-complications are rare, if the albuminuria be excluded, which is an almost constant feature during the height of the fever. That a true nephritis not uncommonly arises is certain from Fraenkel's observations. This authority found that the renal cortex was mainly involved, and that the glomeruli were the seat of hæmorrhagic exudation.

Parotitis is sometimes present and adds a gravity to the outlook, as the gland is prone to suppurate. *Arthritic*

inflammations are occasionally the result of the pneumococcic invasion of the joints.

Diagnosis.—In typical croupous pneumonia a mistake in diagnosis is very unlikely. The high continuous fever rapidly following an abrupt onset, with pain in the side, cough, rusty sputum, and a characteristic pulse and respiration ratio, even before the physical signs appear, are sufficient to settle the diagnosis positively.

Acute phthisis may usually be differentiated by the less abrupt onset, almost always without a chill; but when an apical pneumonia commences without a stormy invasion, and when the rusty sputum is absent, the diagnosis may be impossible till after the expiration of seven or ten days or longer. The discovery of the pneumococcus in the one case and of the tubercle bacillus with elastic fibres in the other may be the only method of settling the diagnosis. Great value always attaches itself to a carefully studied history of the case. In the tubercular lung-lesions liable to be mistaken for apical pneumonia, the history will usually reveal evidence of a degree of ill-health, loss of weight, or slight hæmoptysis before the acute tuberculous invasion of the upper lobe has supervened.

Acute pleurisy with effusion, especially in children, is liable to be mistaken for pneumonia. The differential points are detailed upon page 1389.

Typhoid fever is only likely to be wrongly diagnosed when the careful examination of the chest-signs has been neglected in pneumonia. In a case of latent typhoid fever seen for the first time when a secondary pneumonia is in the act of developing there is sure to be some difficulty. The detection of the characteristic rose-spots and the presence of the Widal reaction will settle the diagnosis even should the pneumococcus be found in the sputum.

Typhus fever, with its abrupt onset and high continuous temperature, closely simulates pneumonia; but the disease is rarely met with except in epidemic form, and the absence of chest signs and symptoms in pneumonia can only last for twenty-four hours, save in the rare cases of the central variety.

Meningitis is often wrongly diagnosed, especially in the pneumonia of children, which is so constantly ushered in by a convulsion, followed by severe headache, vomiting, great restlessness, delirium, and sometimes by retraction of the head. The examination of the chest, which should never be omitted as a routine in all cases of convulsions occurring in childhood, followed by pyrexia, will settle the diagnosis. The meningitis which sometimes occurs as a complication of pneumonia usually only makes its first appearance after the fever has reached its greatest height.

Acute mania is sometimes simulated by a pneumonia which commences with a wild, delirious, and stormy onset which masks the chest symptoms and signs; and *delirium tremens* may be wrongly diagnosed in alcoholic subjects when the immediate cause of the nervous disturbance is a latent pneumonia masked by the restless excitement caused by the illusions.

Appendicitis has been not infrequently suspected, and even operative measures have been undertaken when the abdominal pain and distension have been but the predominant symptoms of an acute pneumonia.

The X-rays, as already mentioned, may prove useful in the demonstration of extensive central solidification of the lung and of the diminution in the diaphragmatic movements. In the elimination of many acute diseases the constant presence of marked leucocytosis in pneumonia is of considerable value and importance.

Prognosis.—The mortality of the disease is high; the percentage varies considerably in different epidemics when pneumonia appears in the epidemic form, and it also varies widely in different countries and at different times. In the British Isles it is somewhere about 15 per cent. in those attacked by the disease, whilst the statistics compiled by Osler show a mortality nearly twice as high in the hospitals of the United States. The hospital-mortality is, however, probably higher in all places than the death-rate observed in private practice, since only the worst cases seek admission.

The prognosis is influenced markedly by *age*. In young children above one year recovery is the rule, whilst in infants and aged patients the mortality reaches as high a figure as 75 or 80 per cent. Aufrecht states that the prognosis is extremely unfavourable in children under five, and conspicuously favourable in those between five and ten years of age.

As already stated, men are more prone to acute pneumonia than women, but of the latter affected a proportionately larger number succumb to the disease. Pregnancy also increases the gravity of the attack.

There is little difference in the prognosis between right- and left-sided pneumonias, though, upon the whole, the prognosis appears to be somewhat more favourable when the disease is upon the left side. It does not appear that apical is necessarily more fatal than basilar pneumonia, though the opposite view is extensively held. The prognosis is obviously more serious in double pneumonia and when the entire lung is involved upon one side.

The patient's previous habits and health are of vital importance in the prognosis, the disease being extremely fatal in chronic drunkards. The presence of such diseases as kidney

and degenerative heart affections greatly adds to the gravity of the outlook ; indeed, the prognosis may be regarded as largely depending upon the soundness of the patient's organs—heart, kidney, and liver especially. The seriousness of the case is also in proportion to the malady which immediately predisposes to the pneumonic attack, as in the pneumonia of influenza, typhoid fever, diabetes, and phthisis.

The individual symptoms influencing the prognosis have been referred to in the general description of the course of the disease. The main point in the prognosis is the condition of the heart ; the rapid degeneration which occurs and ends in death about the seventh day in the majority of the fatal cases does not depend upon the severity of the fever. It is in proportion to the degree of toxæmia, which cannot be measured by the height of the pyrexia or by the extent of the consolidation. Hence the prognosis is considerably influenced by the predominance of cerebral symptoms, and it becomes grave when the typhoid state becomes marked. The signs of failing heart are similar to those witnessed in other conditions : mere frequency counts for less than irregularity and other evidences of weakness. The disappearance of the marked accentuation of the second sound, showing dilatation of the right ventricle caused by the increased resistance in front of it, is a serious sign. Symptoms indicative of poisoning or paralysis of the vaso-motor centres, as evidenced by marked lowering of the general blood-pressure, are always grave.

Meningitis, pericarditis, a purulent pleurisy, obvious abdominal distension, and, more important still, the onset of œdema of the lung, are grave complications ; though, as regards the last-mentioned condition, it must always be remembered that death in pneumonia is seldom directly due to mechanical interference with respiration. Leucocytosis sometimes affords help in making a prognosis when the issue appears doubtful. The complete absence of an increased amount of leucocytes is always a very grave sign, but the opposite condition is not favourable, since a high degree of leucocytosis often is indicative of some complicating purulent collection in the chest or elsewhere.

ATYPICAL PNEUMONIAS.—Little need be said under this heading, since most of the variations from the croupous lobar type have already been incidentally referred to. *Central* pneumonia is a variety of the disease in which the hepatisation commences deep in the centre of a lobe or about the root of the lung, with all the characteristic symptoms and sometimes with rusty sputum, but the pulmonary signs—crepitus, dulness, bronchial breathing, &c.—do not develop for three or four days. The writer has recently witnessed such a case where the typical signs did not appear till the seventh day, though the crisis occurred four days later, and

the patient convalesced in the usual manner, though an acute phthisis was dreaded.

Massive pneumonia is the name given to the rare variety of the croupous form in which all the bronchi in the affected lobe, large and small, are blocked by the fibrinous exudate, the auscultatory signs being masked or absent, a more or less complete dulness on percussion is the only guide to diagnosis, coupled with the occasional coughing up of large arborescent casts of the bronchial tree somewhat like those seen in plastic bronchitis.

Migratory, wandering, or creeping pneumonia is sometimes met with in which the solidification-process slowly spreads or creeps from one portion of the lung to another. The writer has frequently witnessed this form in the aged after an attack of influenza, and in two cases the influenzal bacillus was detected in the sputum. In one of these the typical signs of acute pneumonia appeared in front at the base of the right lung, crept slowly up to the apex, and passed in the opposite direction over the posterior aspect of the same lung, the signs clearing up in one place as they appeared in another.

Pneumonia of childhood has already been referred to as regards its variations from the usual type. Even when certainly due to the specific pneumococcus, it is often liable to take on the broncho-pneumonic or catarrhal type. The onset is usually accompanied by a severe general convulsion; the fever is high, there is no sputum, the upper lobes are affected less frequently than the lower, but much more frequently than in the adult. The delirium and other cerebral symptoms closely simulate meningitis; the gastric disturbances sometimes cause severe and repeated vomiting. The ejecta upon close examination may reveal the characteristic sputa, which had been previously swallowed. A pneumococcic empyema is by no means a rare complication.

A symptom not mentioned in the previous description of typical pneumonia, though it occurs in the adult, is more frequently well marked in the pneumonia of children—viz. the short painful 'grunt' which accompanies expiration.

Pneumonia in the aged often departs from the typical condition in the following particulars. Its onset may be slow and insidious; sometimes it runs an entirely latent course, so that occasionally the condition has not been suspected till sudden collapse from heart-failure draws attention to the physical signs. There may be no fever or sputum throughout, and in the writer's experience the pneumonia is often of the *migrans* type, creeping from lobe to lobe.

Epidemic pneumonia varies considerably in its relation to the ordinary croupous type. In the first place, the marked transmission of the disease from one person to another has been placed beyond doubt in many epidemics; these cases

are probably examples of dual or multiple infection. In some epidemics the clinical picture is dominated by severe cerebral symptoms throughout, in others the *bilious* type is obvious. All the infectious pneumonias as a rule are serious and the mortality is high. The general tendency is towards the development of the typhoid condition, so that the terms *pneumo-typhus* and *typhoid pneumonia* are frequently applied to this variety, which may closely resemble the *asthenic* form met with in alcoholic subjects. These infectious forms will be again referred to under Atypical Pneumonia.

Larval or Ephemeral pneumonia is occasionally seen in the presence of the epidemic form amongst those coming into contact with patients suffering from severe pneumonia. The onset may be abrupt and severe, with high fever, cough, and herpetic eruption, but the attack aborts and rapid convalescence follows after from one to three days without the appearance of any signs of consolidation.

Secondary Pneumonia.—An attack of pneumonia occurring in a patient already suffering from influenza, typhoid fever, plague, cerebro-spinal fever, erysipelas, and diphtheria differs clinically from the true croupous type in many particulars. The symptoms of the primary affection, as might be expected, often entirely mask the onset, and anatomically the progress of the pneumonic process is usually considerably modified in the lung. The tendency in the main may be regarded as towards the bronchopneumonic type of the disease. Though the distribution appears to be lobar, the *post-mortem* changes often demonstrate islands of free pulmonary substance in the midst of the consolidated lower lobe, and the physical signs during life very often enable the physician to be certain that he is not dealing with an honest example of a primary croupous affection involving an entire lobe. This is most obvious in influenzal pneumonia, where over the affected base all the physical signs of the different stages of the pneumonic process may be demonstrated within a comparatively limited area of the chest-walls. The crisis, moreover, seldom is seen in its typical state. Though the modern view is to regard all these pneumonias as the result of the specific pneumococcus, the influence of the other organisms commonly found in the lung and sputum cannot be ignored. The micro-organisms of the primary disease are often found not only associated with the pneumococcus, but streptococci, staphylococci, and the bacillus coli have been also frequently found.

Under the title of *Atypical Pneumonia*, Aufrecht, Friedreich, Kühn, and others describe the several varieties of pneumonia already mentioned under 'Epidemic Pneumonia,' and which are more or less markedly infectious. These authorities maintain, amongst other reasons, that as typical croupous pneumonia is never transmitted from one person to another,

the epidemic form of the disease should be placed in an entirely different category. The identification of the different organisms, however, has not been carried out with sufficient precision to justify a distinct anatomic basis for such a classification. It is true that the morbid changes in the main resemble those found in the croupous type; but grey hepatisation is seldom so marked; the section of the lung is frequently less distinctly granular, and, though the distribution of the pneumonic process is lobar, there are constantly found areas of unaffected pulmonary tissue scattered throughout the affected lobe.

In the opinion of the writer, who has chanced to observe several of these atypical pneumonias, though clinically they depart considerably from the croupous type, they do not vary from it to a greater degree than does a genuine pneumococcic pneumonia occurring in a typhoid or influenzal patient. The most frequent characteristic of the latter, in addition to the points already detailed, is the tendency of the pneumonic process to follow the migratory or wandering method of extension.

This objection to a rigid classification is strengthened by the great variations from the true croupous type which are observed in the *terminal pneumonias* met with in diabetes, Bright's disease, chronic phthisis, and arterial sclerosis, in all of which the specific pneumococcus may be found. If the infectious or epidemic varieties are to be placed in a different class from the croupous, so also should the secondary, the asthenic, wandering, and typhoid varieties be also so isolated. It is probable that a classification of pneumonia upon a more rigidly scientific basis will be an event in the near future.

Treatment.—At the present time this subject is in a very unsettled and unsatisfactory state, widely divergent views being held, some writers maintaining that the greatest hope of success lies in a purely expectant line of treatment; others place their faith in various so-called specifics; whilst the great majority adopt the plan of treating the symptoms of the disease as they rise. Upon pages 760-767 in the author's 'Dictionary of Treatment' (4th ed.), the details of the different methods are discussed; only general principles need be here briefly referred to. Two important facts must not be lost sight of—viz. that the disease is a self-limited one, running a short course, in the great majority of cases of not more than seven or nine days, and that it tends to produce a fatal result by the depression of the cardiac muscle through the action of the toxins manufactured by the pneumococcus.

No agent is known which can be regarded as capable of exercising any specific action upon the pneumococcus or its toxins.

The result of the treatment of the disease by the application of the principles of serum-therapy holds out little prospect of success, since the pneumococcus appears to be liable to extraordinary variations in its developmental characters in different individuals. Hence, as in erysipelas serum-therapy, the different strains of the microbe used in the manufacture of the serum are only of value when the latter is injected into the tissues of a patient who chances to be suffering from the precise variety or strain of the pneumococcus used for the manufacture of the serum. To overcome this difficulty a *polyvalent serum*, prepared by using cultures of various cocci, has been prepared, but the results cannot be regarded as satisfactory. The treatment based upon the principle of raising the opsonic index by stimulating phagocytosis is still in its infancy as regards its application to pneumonia.

The toxæmia therefore can only be minimised by the employment of such agents as will hasten the elimination of the poison from the body. General blood-letting in former days certainly effected the removal of a considerable amount of the poison in the circulatory fluid extracted from the opened vein, but the method is not without its serious depressing drawbacks. Where venesection is undertaken, a larger amount of normal saline solution should be injected hypodermically, by which means the quantity of blood removed without risk can be greatly augmented and the operation can be repeated. Diaphoretics, diuretics, and cathartics are employed sometimes with much advantage upon the same principle. By the use of these depletory measures it is claimed that the toxæmia in the early stage of the disease may be so lessened that serious cardiac degeneration is prevented. The advantage of large hypodermic or rectal injections of saline solution, independent of blood-letting, is beyond doubt.

There seems, however, an approach to unanimity upon the necessity of anticipating the heart-failure, and hence digitalis is adopted by many as a routine agent from the beginning of the attack. Its value as a cardiac tonic is, however, very doubtful in all diseased conditions associated with a high degree of fever. Much more reliable is the action of strychnine, which should be always given by the mouth in small doses from the early stage, so that when signs of heart-failure manifest themselves the drug may be boldly pushed by the hypodermic route. This is the only reliable means of combating serious cardiac failure in such conditions as exist in pneumonia, but the dose must be not less than $\frac{1}{20}$ gr. Many authorities combine digitalin with the hypodermic dose of strychnine, believing that, though useless by the mouth, digitalis is valuable when given by the hypodermic route. Brunton, who first pointed out the value of

strychnine in the heart-failure of pneumonia, combines it with oxygen-inhalations, and many authorities regard the action of these inhalations as of much greater importance than mere antidotal remedies for the relief of cyanosis and dyspnoea, since they consider that the saturation of the blood with oxygen markedly reduces the toxæmia. The gas should never be administered by a mask, as in chloroform-inhalation, but it must be given largely diluted with atmospheric air.

Brunton has more recently emphasised the great value of calcium chloride as a heart-tonic in pneumonia; but before pushing the drug it will be well to test the coagulability of the blood by estimating the time of clotting of a drop removed by a needle-prick. This precaution seems necessary when the large amount of fibrin present in the blood of the pneumonic patient and the tendency towards cardiac thrombosis are considered.

Alcohol as a routine agent is passing into well-merited disfavour, but its value as a diffusible stimulant after serious heart-failure has suddenly appeared is beyond question in such an emergency.

The general management of the patient as regards diet and hygienic considerations is to be conducted upon the same lines as in any other feverish condition. The relief of the prominent symptoms is to be attempted upon recognised therapeutic principles; thus the pain should be relieved by hot poultices or iced applications, according to the sensations of the patient. When severe, morphia by hypodermic injection may be safely used, but in the late stages of the disease this drug should be employed with the greatest caution, as unquestionably, even in a moderate dose, it may cause suddenly fatal collapse, as pointed out by Sir William Gairdner.

The pyrexia may be met by quinine in large doses, and Aufrecht maintains that full hypodermic doses of this drug have a powerful effect in antagonising the deleterious action of the toxins, even when they produce but a slight impression upon the fever. When the temperature reaches a high figure, and in all cases where hyperpyrexia has set in or is threatened, nothing but the cold bath or continuous cold sponging should be relied upon.

PNEUMONIA, CATARRHAL

This condition, which is variously described as Lobular Pneumonia, Catarrhal Pneumonia, Vesicular or Disseminated Pneumonia, and Capillary Bronchitis, is dealt with under 'Bronchitis.' It is essentially a catarrhal inflammation, originating in the mucous membrane of the bronchi, spreading

backwards and invading the terminal bronchioles which end in the alveoli. The disease is mainly one of childhood, being very rarely seen in the adult, though occasionally it may be observed in the aged. In its most typical form it is found accompanying the bronchitis of measles and whooping-cough and sometimes typhoid fever and rickets.

Its clinical relationship to lobar or croupous pneumonia is only of importance in those cases where a series of lobules become involved in such numbers as by their coalescence to simulate a lobar attack, in which case the signs may be almost identical, the consonating râles becoming so fine as to simulate the crepitant râle of Laennec and the area of consolidation affording the usual dullness, bronchial or tubular breathing, bronchophony, &c. The distribution of the consolidation, however, is usually sufficient to differentiate the lobular inflammation, which rarely, if ever, is limited by the true lobar boundaries, being commonly found about the angle of the scapula or the mid-dorsal region, and always associated with scattered islets of partial consolidation in the opposite lung. The fever seldom or never reaches the high degree seen in croupous pneumonia, and it is markedly irregular in type; there is no distinct crisis, the lung-signs are the product of a slow development, and pass off as a rule gradually. The most obvious clinical symptom is the severity of the dyspnœa and cyanosis, which are the result of secondary pulmonary collapse; rusty sputum is almost invariably absent. The above features resemble closely those sometimes observed in the pneumonia of adults suffering from influenza, which, according to some authorities, is but a form of the catarrhal variety.

The history of the case and its subsequent progress seldom leave any doubt about the nature of the catarrhal or lobar affection. Though the pneumococcus has often been found present in the alveolar contents, pathologically the catarrhal form must be regarded as distinctly different from the true croupous affection.

ASPIRATION or DEGLUTITION PNEUMONIA

This is the name given to a form of pulmonary inflammation which, though in some respects differing from catarrhal pneumonia, exhibits similar naked-eye changes in the lung and closely allied clinical symptoms and signs.

Etiology.—This condition is produced by the aspiration of infectious organisms from the mouth or bronchi which reach the alveoli during comatose conditions. Thus during the insensibility following apoplexy there is a copious outpouring of bronchial mucus, which in the expiratory process and by the aid of the cilia finds its way into the mouth. After the admixture of this thin secretion with the buccal mucus

and saliva it is drawn downwards or aspirated during inspiration through the paralysed larynx, bringing with it various micro-organisms, which upon reaching the bronchioles find their way into the alveoli, where they excite a severe inflammation of the air-cells and inter-alveolar tissue. Aufrecht maintains that the condition is identical with that experimentally produced in animals by section of the vagi, and that it differs from ordinary catarrhal pneumonia by the complete absence of any inflammatory involvement of the bronchioles.

The term *deglutition-pneumonia* is used where, in other paralysed conditions of the larynx, particles of food are aspirated and produce the same effect; the term should be discarded. It is a common terminal event in bulbar paralysis.

Drowning produces similar results, and the writer has observed several cases where pulmonary abscesses have followed the aspiration-pneumonia produced by submersion in the sewer-laden contents of the water in the vicinity of the public docks and harbour.

Ether-inhalation is liable to eventuate in an aspiration-pneumonia practically identical with that observed in apoplexy, uræmia, and other comatose conditions in which the septic contents of the mouth are mixed with the bronchial secretion and sucked backwards towards the alveoli, the condition being recognised as *ether-pneumonia*.

Hæmoptysis from whatever cause may be the etiological factor, and in an important group of cases the affection originates in the aspiration of septic products formed in bronchial ulceration, bronchiectatic and phthisical cavities, and sometimes in perforating empyemas, and in malignant disease of the œsophagus or mediastinum, where septic products perforate the respiratory tract.

The disease is common after operations for the removal of the tongue, maxillæ, larynx, nasal bones, &c., and after tracheotomy. It is also especially liable to supervene in diphtheria, and is a portion of the process in acute tuberculosis when a shower of bacilli from some latent focus in the diseased lung finds its way into the bronchial tract to be aspirated into the alveoli of the healthy lung and into unaffected parts of the originally diseased organ.

The writer has once observed its occurrence after alcoholic and opium poisoning, and it has been reported in carbonic-acid, coal-gas, and carbolic-acid narcosis. Sometimes it has been caused in the newly born babe by aspiration of septic vaginal discharges during the late stage of delayed delivery.

Symptoms and Signs.—These, in the main, correspond with those of a catarrhal pneumonia, save that the most dependent portions of the lung are invariably the site of the disease, which is bilateral as a rule. The constitutional symptoms vary with the virulence of the organisms aspirated,

the sputum becoming fetid and gangrenous in odour when the inflammation ends in the death of the inflamed pulmonary tissue. In the tubercular variety of aspiration-pneumonia a more or less profuse hæmoptysis usually marks the advent of the perforation, as pointed out by Bäumer.

Diagnosis.—This is as a rule easy, the ordinary catarrhal pneumonia being associated with its exanthematous or whooping-cough affection when not occurring as a primary capillary bronchitis; moreover, it is a disease limited practically to the period of childhood. The presence of the etiological factors already detailed is never wanting in the history of the aspiration variety of pneumonia.

Prognosis.—This will entirely depend upon the nature of the organisms gaining admission to the alveoli. In tubercular and malignant cases the prognosis is always certain. The outlook after prolonged submersion in contaminated water, septic sewers, or cesspools is obviously grave. Probably in the aspiration-pneumonia following ether-inhalation and other forms of temporary insensibility the prognosis may be gauged by the state of oral sepsis and, to some extent, by the duration of the period of insensibility and its profoundness.

Treatment.—Little can be done save to minimise the amount of septic material left in the lung in cases of submersion by inversion of the body before artificial respiration be vigorously commenced. The fact that the ordinary septic organisms normally present in the mouth and in the inspired air never under ordinary conditions reach the alveoli of the lung shows how little need be expected from the inhalation of antiseptic sprays, as these certainly do not penetrate beyond the small bronchi. The writer recommends the routine practice of administering a speedy emetic as soon as possible after the immediate recovery from submersion in sewer-contaminated water, with the view of getting rid of all fluid accumulation in the finer bronchioles. The inhalation of oxygen may be valuable in some cases.

PNEUMONIA, CHRONIC

The consideration of the chronic inflammatory process met with in the pulmonary tissue is involved in great difficulty and confusion owing to the multiplicity of the causes which induce it. The disease is known as Chronic Interstitial Pneumonia, Cirrhosis of the Lung, and Pulmonary Sclerosis, and also, owing to the frequency with which tuberculosis is associated with it, it is often described as Fibroid Phthisis. In the great majority of cases the condition is held to be secondary to a tubercular infiltration, but there can be little doubt that not infrequently tubercle invades a lung which

has been the site of some chronic interstitial change induced by the inhalation of an atmosphere laden with particles of coal, steel, cotton, linen, or stone dust, and pulmonary cirrhosis can undoubtedly exist without tuberculosis.

Etiology.—Any irritant, as tubercle and the foreign substances just mentioned, which cause the condition recognised as *pneumonokoniosis*, may be the starting-point of the chronic inflammatory process.

The bronchopneumonia which occurs most frequently in children as a sequel or complication of measles and pertussis may sometimes be seen to eventuate in a chronic interstitial pneumonic process, starting in a fibroid proliferation originating in the bronchiectatic dilatations.

Chronic pleurisy is the starting-point in some cases, the hyperplasia of the pleural fibrous tissue steadily invading the interstitial connective tissue of the lung.

There is a well-recognised form of the disease seen in rare cases where a chronic interstitial pneumonia follows a typical croupous lobar attack. This, instead of resolving at the usual time, passes slowly into a cirrhosis of the lung, caused by a new growth of connective tissue arising in the alveoli.

The syphilitic, like the tubercular process, may manifest itself by an extensive proliferation which originates in the peribronchial connective tissue.

It will thus be obvious that a chronic interstitial pneumonia may arise from any diseased action originating within the alveoli, the interlobular fibrous tissue, the bronchi, or the pleural membrane. The etiological factors therefore are as numerous as are those various agents which cause destruction of the pulmonary tissue.

With these must also be included those mechanical impediments to the circulation, as valvular lesions, adherent pericardium, &c., which eventuate in a chronic engorgement of the lung-vessels.

Men are more frequently affected than women, and, though not necessarily associated with hepatic cirrhosis, an alcoholic history is often obtainable. The disease may occur at any age, but is seldom seen under twenty.

Morbid Anatomy.—The disease is usually limited to one lung or to a lobe of the lung. In the rarer bronchopneumonic form which follows measles and whooping-cough it may exist in scattered islets throughout both organs, though even in such cases the tendency is towards a coalescence of these indurated masses with their enclosed dilated bronchi, so that a more or less lobar character is given to the cirrhosis. The lower or middle lobes are most frequently involved; the naked-eye appearances are caused by firm bands of whitish or grey fibrous tissue, which may be traced from the root of the lung into the intralobular septa, spreading along the

bronchi and vessels. The pressure of these dense cords or partitions of connective-tissue growth compresses the pulmonary tissue, which becomes airless, almost black in colour, and so firm that it may cut like a piece of cartilage. The bronchi are dilated and show on section as numerous cavities whose walls are held open by the cicatrising fibrous tissue which envelops them. The dark surrounding collapsed pulmonary parenchyma, contrasting with the lighter bands of connective tissue, gives the section a distinct marbled or mottled appearance.

The fibrous bands are usually much thicker and extensive in those cases of cirrhosis which have originated in chronic pleural inflammation, and their cords can be easily traced inwards from the surface of the lung as they penetrate deeply between the lobes. In the primary tubercular cases the process may be traced from the apical region downwards, and generally dense pleural adhesions are found surrounding an old phthisical cavity with thickened walls. Much emphysema is commonly present in the unaffected lung and in patches scattered throughout the cirrhotic organ. Microscopic examination usually reveals some thickening of the alveolar walls, obliteration of the capillaries, and changes in the epithelial lining of the air-vesicles, which are probably the secondary results of irritation and compression by the newly formed fibrous tissue.

As in cirrhosis of the liver, with which the pulmonary sclerosis is closely allied from the pathological standpoint, the final tendency of the new-growth towards cicatricial contraction shows itself in the very marked diminution of the bulk of the affected lung. The shrunken organ may be at first invisible upon opening the thorax, when bound down and concealed by dense pleural adhesions by the side of the spinal column, the enlarged and emphysematous lung having crossed over the middle line and occupied a considerable part of the space formerly occupied by the diseased organ. The heart is considerably displaced towards the affected side, and its right ventricle generally greatly hypertrophied. The chest-wall has fallen in and the shoulder upon the affected side is much depressed, and often the spinal column is deformed.

Symptoms. — The patient is usually past middle-life, the disease being rare in childhood. Commonly, when first coming under observation there is a history of increasing shortness of breath or of dyspnoea and cough upon exertion, which may have existed for many years. The most obvious clinical feature is the extreme chronicity of the disease; and not infrequently the cough and dyspnoea have been of such gradual onset that the patient is scarcely conscious of his discomfort unless upon active exertion, though his fingertips may be markedly clubbed and other signs are present

which demonstrate a long-standing degree of serious pulmonary embarrassment. There is usually little or no fever, and the general nutrition may be for a long time fairly well maintained.

Hæmoptysis is often present even when there is no evidence of tuberculosis. The cough and expectoration are in proportion to the amount of bronchiectasis accompanying the fibroid changes in the lung. When this is marked, the clinical picture is that seen in bronchial dilatation, the cough being severe and paroxysmal and often associated with profuse purulent and sometimes with fetid expectoration. A temporary retention of the decomposing purulent contents of the bronchial cavities may cause septic or hectic symptoms at times which closely simulate the clinical picture of a chronic phthisis, especially when hæmoptysis happens to be also present.

Physical Signs.—These are obviously of much greater value and importance than the symptoms, which are often very vague or indefinite.

Inspection.—The affected side is contracted and moves little during respiration; the intercostal spaces are encroached upon or obliterated by the overlapping ribs. The shoulder is depressed, the angle of the scapula projects, and there is usually some lateral spinal curvature, the concavity of the column looking towards the affected side. The cardiac impulse is visible in its abnormal position, being dragged towards the affected lung; and in left-sided cases it may be elevated or a considerable proportion of the heart's surface may be denuded of overlapping lung, so that pulsation is seen over a wide area of the thoracic wall.

Mensuration.—The cyrtometer will reveal a very distinct diminution in the measurement of the affected side, and the opposite side is larger than the normal.

Palpation confirms the obvious diminution of the respiratory movements and reveals the absence of vocal fremitus when there is great pleural thickening, which also may be detected by the increased resistance, unless the spaces are so obliterated that this sign cannot be applied. In the uncommon cases where the pleura is normal the vocal fremitus is increased.

Percussion generally reveals dulness owing to the great pleural thickening; marked increase in resonance may be elicited over large bronchial dilatations. A valuable sign in this connection is the tympanitic note, which may replace complete dulness after the patient has been seized with a severe paroxysm of coughing which has pumped out a large bronchiectatic cavity in the base of the lung. A similar result may occasionally be obtained by a marked alteration of posture, which sometimes may empty a bronchial dilatation

without inducing coughing. The opposite side of the chest is always hyper-resonant.

Auscultation gives very variable results. When the stethoscope is applied over a bronchial dilatation the signs of cavernous breathing may be marked, but usually these are obscured by râles and rhonchi, which may, however, have a metallic character added on. Metallic gurgling is often heard upon coughing, especially about the middle of the chest or below the apex. Of more importance, however, is the absence of the true vesicular murmur over the affected lung; this, at the apex, is often replaced by breathing approaching the bronchial in type, whilst over the base the breath-sounds are usually weak, distant, or indistinct, even when not obscured by the râles and rhonchi which are commonly present. Bronchophony is seldom well marked owing to the thickness of the pleura. Various creaking friction-sounds are usually audible in auscultating at some distance from the main site of the cirrhotic lobe.

The heart-sounds may locate the displacement of the organ in the absence of a visible or palpable apex-beat; usually there is evidence of increased peripheral resistance in the pulmonary circulation, as seen in the accentuation of the second sound at the base of the heart. A tricuspid systolic murmur may be audible when the compensatory hypertrophy has commenced to break down in the later stages of the disease, in which case dyspnoea and anasarca will be present.

Life may be prolonged for twenty or thirty years or more, the disease showing little direct tendency towards a fatal termination unless complicated by an attack of acute bronchitis, croupous pneumonia, or other serious illness. Sometimes, from prolonged pus-formation in the dilated bronchi, amyloid degeneration of the liver, spleen, kidney, and intestinal mucous membrane supervenes, and proves fatal by exhausting the patient's strength through uncontrollable diarrhoea. Occasionally death results from dilatation of the right chambers of the heart setting in after a comparatively mild attack of bronchial catarrh or influenzal infection.

Diagnosis.—The established condition, when taken in conjunction with the very chronic history of the case, as a rule leaves little doubt about the nature of the disease, if two other affections can be eliminated: these are *chronic tubercular phthisis* and *perforative empyema*.

In *phthisis* the changes may be practically identical, and even a naked-eye examination of the organs, when removed from the body, may fail to distinguish between the two conditions. The great majority of all examples of cirrhosis of the lung are tubercular, and the only pathognomonic sign of their true nature exists in the presence of the tubercle bacillus in the sputum or pulmonary tissue. Even when this is forth-

coming, the doubt may reasonably arise that the case was one of primary pulmonary sclerosis upon which, in its later stage, tuberculosis had been engrafted; though the generally accepted view is to regard such cases as being of an unusually slow type of phthisis from the beginning.

The following considerations will assist in arriving at an accurate diagnosis. In phthisis of very chronic type, not only are tubercular changes nearly always present in both lungs, but evidence of tubercle may be found, if sought for, in the larynx, bones, lymphatic glands, genito-urinary tract, or peritoneum. Primary pulmonary cirrhosis is, upon the other hand, nearly always confined to one lung or to one lobe. Where the bacillus cannot be detected in the sputum the tubercular diagnosis may be made by the reactionary fever following tuberculin-injections; but this method is obviously only applicable in apyrexial cases.

Chronic empyema is not likely to be mistaken for cirrhosis, unless perforation of a bronchus has occurred, in which case the thoracic signs and pulmonary symptoms are practically identical, the expectoration closely resembling that discharged paroxysmally from one large or a series of moderate-sized bronchiectatic cavities. The history of the origin and progress of such a case, when carefully scrutinised, should never lead to much doubt, as it affords even more reliable evidence than that obtainable by exploratory puncture, since the needle, after perforating a greatly thickened pleura, may in one case be made to enter the pleural sac, or, in the other, to penetrate a large bronchial cavity.

Malignant disease in the thorax should never be a source of doubt in the diagnosis, since the essential clinical feature of pulmonary cirrhosis is its extreme chronicity, which always outlasts the longest possible duration of the slowest progress of any malignant process.

Prognosis.—The chief points in connection with the forecast have been incidentally referred to in the description of the origin and progress of the disease. In a given case, though it can be safely prophesied that the affection is an incurable one, there may be conditions present which enable the physician to predict that life may be prolonged till even an advanced age. These may be observed in the state of the general nutrition and physique of the patient, but especially in his environment and the facilities which he possesses for living a life free from strain and exposure to fogs, damp, and cold, and depressing vicissitudes of all kinds. Thus, if his means will permit of his living an open-air life in winter in a suitable climate, his outlook is decidedly more favourable than that of the less fortunate victims of the disease.

The prognosis is, upon the other hand, always grave in the presence of any cardiac or renal disease, and obviously it

deepens upon the discovery of the tubercle bacillus in the sputum.

Treatment.—As the cirrhotic process is beyond the influence of drugs, the management of the case resolves itself into the relief of symptoms and the maintenance of the general nutrition in the highest degree possible. This latter is carried out upon the hygienic principles indicated in the treatment of phthisis, as open-air existence and forced feeding ; hence the importance of the patient (when his means permit) seeking a mild equable climate in which all his time can be spent in the open air. The treatment of symptoms is mainly confined to the relief of cough and expectoration ; but no more objectionable plan can be conceived than the one too often followed—viz. the keeping of the patient drenched with sedatives and nauseating expectorants with the view of stopping the cough. A narcotic is seldom, if ever, indicated in this condition for the purpose of allaying unnecessary coughing. The ideal, as in all other chronic pulmonary and bronchial diseases, should be to act upon the bronchial surface so as to liquefy the sputum and render it more easily pumped up. The best drug for this purpose is the iodide of sodium, combined with small doses of strychnine. Another indication of vital importance is the rendering of the sputum as near to the aseptic condition as possible when there is a marked degree of bronchiectasis. Volatile substances, as eucalyptus oil, creosote, turpentine, &c., used internally and by inhalation, certainly tend to prevent the multiplication of the pyogenic and putrefactive organisms which flourish in the old bronchial dilatations. Cod-liver oil is also indicated in nearly every case.

PNEUMONIA, DESQUAMATIVE

This is the name given by Buhl to a form of pneumonia whose onset resembles that of the croupous variety, with rigors, high fever, slight cough, rusty sputum, &c. The upper lobes are most frequently involved, and the physical signs are practically identical with those observed in croupous pneumonia, but the progress of the disease is slow and variable. The essential pathological feature is the distension of the alveoli, with large swollen cells and giant-cells containing several nuclei ; these are the desquamated alveolar epithelium, which is shed in abundance as the disease progresses. The character of the sputum is said to be pathognomonic, consisting almost entirely of desquamated alveolar epithelium in various stages of granulation and fatty degeneration. Aufrecht, whilst acknowledging the existence of such a condition as a rare primary disease, maintains that most of the cases are those of a true secondary desquamative pneu-

monia originating in connection with more or less latent tubercular foci in the lung. This explains the facts that the majority of the cases follow upon a severe hæmoptysis and that the upper lobe is the usual site of the disease. These secondary cases can be distinguished, however, from acute tuberculosis by the massive nature of the consolidation, which often exists without cough and fever or other constitutional symptoms.

The importance of the subject is obvious, as the recognition of a desquamative type of pneumonia which tends to end in a slow resolution affords an explanation of many cases of apical pneumonia of several weeks' duration which have been hitherto regarded as examples of the croupous variety, with delayed resolution.

In the secondary form of the disease, after the disappearance of all signs of consolidation, sometimes evidence of the primary latent tubercular focus may be observed. As insisted upon by Aufrecht, the conception of a secondary desquamative pneumonia fully explains the hitherto puzzling phenomena observed in many cases of latent phthisis where extensive transitory consolidation of an upper lobe has been preceded by a smart and unexpected hæmoptysis. Though the consolidation may leave the pulmonary tissue intact, and even permanent recovery may occasionally ensue where tubercle bacilli have been detected in the sputum, the general tendency of such attacks is to eventuate in further disintegration of the lung. Some examples of supposed acute croupous pneumonia which pass into the condition recognised as chronic pneumonia are probably of this primary desquamative type.

PNEUMONIA, HYPOSTATIC

This is the term applied to the form of pulmonary engorgement which affects the bases of both lungs in bed-ridden patients under certain conditions.

Etiology and Pathology.—The essential cause is always some feebleness of the ventricular contractions associated with loss of tone in the vascular walls. On account of this combination of circumstances, the circulation in the lungs is unable to overcome the influence of gravity, so that the blood accumulates in the most dependent portions of both organs.

All adynamic conditions, especially in the aged, are liable to be associated with this form of pulmonary congestion after the patient has been compelled to take to bed. Thus it is very common in the surgical hospital-wards amongst debilitated patients whose injuries compel them to remain long in the horizontal posture. It is constantly observed in the

medical wards, setting in soon after cerebral apoplexy and severe spinal lesions, and it is seldom absent in very protracted cases of enteric fever, except in the very young.

In coma, from whatever cause, it may be found, and it is then usually associated with aspiration-pneumonia. When the decubitus is not upon the back the lower lung may alone suffer, but usually the opposite is affected also, though in lesser degree.

In the first stages the affection is mainly, if not entirely, due to engorgement of the pulmonary vessels, to which the usual term *hypostatic congestion* is strictly applicable. The lung if seen in this stage is of a deep red at its most dependent portions, resembling the organ in the first stage of croupous pneumonia; but the toughened, almost airless, and sodden portions involved do not correspond with the lobar distribution of that disease. Upon section, blood and serum ooze from the cut surface. At a later stage the congestion passes into a distinctly pneumonic process, though the affected tissue does not entirely sink in water like the croupous hepatisation; it is dense and does not float upon the surface, and blood ceases to pour from its freshly cut surfaces. The alveolar epithelium is degenerated; a coagulation-necrosis involves the contents of the air-cells, which may also exhibit fibrinous exudation.

The term *splenisation* conveys an accurate idea of the characteristics of the airless lung in the stage of hypostatic pneumonia. There is always a corresponding hyperæmia or engorgement observable in the bronchial mucous membrane of the affected portions of the lung, but as a rule the pleural membrane shows little change.

Symptoms.—These may be entirely absent, the condition being often found in a routine examination of the chest of a bed-ridden patient who presents neither cough, dyspnœa, nor fever.

The physical signs are often vague. Some diminished movement may be detected, but dulness can always be elicited over the most dependent portions of both lungs, often as high as the middle of the scapula. The note is never quite flat, as in effusion, but exhibits some tympanitic quality. The breath-sounds are feeble and distant, and have lost their vesicular character; true bronchial breathing and bronchophony are very rarely heard, though the feeble breath-sounds may have a blowing character. Râles are frequently present; these are of variable degrees of fineness or coarseness, according to the size of the bronchial tubes involved in the hyperæmia.

Sometimes the sputum, which is usually scanty, may be tinged with a faint streak of blood; but true hæmoptysis does not occur, and not infrequently expectoration is absent throughout.

The affection is too often overlooked, but the knowledge that the condition is liable to supervene should render periodical routine physical exploration of the chest a necessity under the circumstances already narrated.

The prognosis is that of the primary affection, and the condition of the myocardium is an important consideration in the prognoses of those cases where an obviously fatal termination is not necessarily to be expected from the primary disease.

Treatment.—Whilst in the main the management of the case resolves itself into that of the primary disease, much may be done in the prevention or minimisation of the condition by a close and persevering attention to the posture of the patient. This should be changed as frequently as his primary ailment will permit, and even when compelled to remain upon his back he should be encouraged to make deep inspiratory efforts at regular intervals.

Cardiac tonics and stimulants are indicated: strychnine hypodermically is much more to be depended upon than digitalis by the mouth. When cyanosis and dyspnoea set in, oxygen-inhalations will be valuable.

PNEUMONOKONIOSIS

This is the name given by Zenker to the chronic pneumonic process induced by the inhalation of air laden with particles of dust of various kinds. The condition has received a variety of titles indicative of the nature of the inhaled foreign particles. Where coal or carbon dust is the agent, as in miners and coal-carriers, the disease is known as *Anthracosis*. *Siderosis* is the name given to the condition produced by breathing air laden with fine particles of metal, as steel, brass, oxide of iron, &c., as seen in needle and file makers, metal-grinders, and iron-workers. *Chalicosis* or *Silicosis* is the corresponding condition produced by the inhalation of mineral or stone dust, as met with in stone-cutters, knife-grinders, and makers of mill-stones and grinding-stones, &c.

Anthracosis has been the subject of a vast amount of literature and of much experimental research, and unquestionably the last word has not been said upon its most controversial aspects. Only the briefest outline is here admissible, as the subject is of much more interest from a physiological or pathological than from a clinical standpoint, since it is universally accepted that the deposition of coal-dust or soot in the lung-tissue usually produces very little irritation, and it is a well-recognised fact that miners are much less prone to pulmonary tuberculosis than are other artisans, the

anthracosis being believed to confer a certain degree of immunity against phthisis.

The interest centres round the point of how the coal-dust gains admission to the lung. In the first place, the pathological changes are unquestionably obvious—the dust is found extensively deposited in the pulmonary tissue. It is almost absent or found only in the most sparse amount in the alveoli of subjects who have long lived in an atmosphere laden with the dust-particles, and this is also true of the dust-free condition of the alveoli in animals caused to breathe such an atmosphere. The main lines of deposit are along the peribronchial and perivascular tissue and between the interlobular connective fibres. It is also constantly observed under and within the interstices of the pleural membrane, in all of which cases, according to the majority of pathologists, the carbon-particles are free and not enclosed in cells.

These facts led many observers to believe that the dark or black coloration was due to pigmentation-products derived from the blood, and not to carbon-deposition, but the carboniferous nature of the deposit is now unquestioned. During life the sputum of persons breathing coal-dust is loaded with carbon-particles; these exist in two forms—viz. as free particles and as particles enclosed in cells, which seem to be desquamated alveolar epithelial cells; they are also found in the ordinary mucus-corpuses, which, acting as phagocytes, engulf the foreign matter as it adheres to the moist bronchial lining—a natural provision to prevent their entrance into the air-cells. As regards the particles enveloped in the alveolar epithelial cells, it is supposed by many that they have been engulfed by the cells after these have been shed from their normal site. This latter view is accepted by those who maintain that foreign particles in the inhaled air are never permitted to reach the alveoli, and recent researches appear to establish this belief. Many observers, including Buhl, Arnold, Ins, and Schottelius, affirm that the carbon-particles found in the pulmonary stroma, pleura, and bronchial glands find their way there after being first enclosed in the alveolar epithelium or white blood-corpuses, afterwards travelling by the lymph-nodules, lymph-stream, or tissue-juice. Aufrecht maintains, upon the other hand, that free carbon-particles enter the pulmonary tissue through the smaller bronchial walls by separating the longitudinal muscular fibres, their penetrative power being explained by the negative atmospheric pressure existing in the thorax during inspiration, which draws or sucks-in the free particles through the distended layers of the musculature of the wall of the fine bronchi. This authority emphasises the fact that many of the particles found in the pulmonary tissue are much larger than the cells which have been theoretically held to convey them thither.

It has been long known that carbon-particles in anthracosis or coal-miner's disease have been found in abundance in the liver and spleen, and Villaret maintained that the coal-dust in the lung was brought to the pulmonary tissue from the digestive tract *via* the portal vein, and not by inhalation.

The subject has been quite recently re-investigated by Calmette and Guérin, and their results are of enormous importance, since they have a direct bearing upon the spread of tuberculosis in the lung. The matter has been already referred to under the etiology of phthisis (p. 1301). These observers found that, contrary to the accepted view, anthracosis cannot be produced in animals by making them inhale an atmosphere laden with soot-dust, if the precaution be taken to prevent the entrance of the carbon-particles into the stomach by plugging the œsophagus. The carbon-particles, like the bacilli of tubercle, according to these observers, reach the lung after their arrest in the mesenteric glands, into which they gain access through the intestinal epithelium. In the chyliiferous channels they are seized upon by the polynuclear phagocytes, and eventually gain admission into the venous system, to be deposited in the bronchial glands and the lung.

In the lungs of miners the carbon may be so abundant as to produce a uniform black colour, and the cut surface often exudes a black inky juice which stains the fingers. In most instances the organs are otherwise quite normal, and crepitate upon pressure, no fibrous proliferation or other evidence of a chronic pneumonic process being visible to the naked eye or microscope. Occasionally the deposit is limited to dense black patches, and this is very striking in the rather uncommon cases with which tuberculosis is associated. In cases of very long standing, in addition to the deposit of carbon found in the interlobular, peribronchial, and perivascular tissue, and in the tracheal and bronchial glands, as already mentioned, there are evidences of some light degree of fibroid proliferation, such as is seen in ordinary chronic interstitial pneumonia. Very rarely are any necrotic areas observed in the lung; but sometimes, even where tubercle could not be detected, small abscess-cavities have been found in communication with a bronchus, these being situated in the middle of a dense patch of cirrhotic lung-tissue. More or less bronchial change is often present, the mucous membrane exhibiting the inflammatory thickening characteristic of chronic bronchitis.

Whilst it is thus obvious that a high degree of anthracosis usually exists without leading to any fibroid changes, or to but the most trivial increase in the connective-tissue elements in the lung, it is different in siderosis and chalicosis. Iron-dust and fine particles of stone excite a marked degree of

interstitial pneumonia, the lungs being found studded with small nodules, which grate against the knife in cutting. These may reach the size of a large pea, and consist of airless pulmonary tissue, in which the foreign matter is deposited, surrounded by firm and dense bands of cicatricial tissue. The nodules are usually greyish-white in the middle, and sometimes may exhibit a small central opening. The chronic pneumonic lungs of iron-workers may be almost as dark as those of miners, but when iron-oxide is the cause they become brick-red in colour.

The supervention of tuberculosis is a common event, and tubercular nodules, caseating foci, or suppurating phthisical cavities are frequently observed, associated almost always with more or less pulmonary emphysema, caused by the chronic bronchitis accompanying the disease.

In the lungs of artisans engaged in the preparation of flax for the spinning of yarn for linen-manufacture, the main changes observable are those of a high degree of emphysema, caused by the dry catarrh induced by breathing the fine soft dust which permeates the air of the preparing and hackling rooms.

Symptoms.—The coal-miner usually escapes serious pulmonary trouble and only suffers after many years of exposure, though he has commonly some black sputum. When severe bronchial irritation eventually supervenes, the expectoration becomes profuse, purulent, and it may be inky black. Under the microscope abundance of alveolar epithelial cells are seen, with particles of carbon in their interior. The symptoms and signs are those of a chronic bronchitis and emphysema, which cause considerable shortness of breath and wheezing. When phthisis has supervened, large particles of coal-dust may appear in the sputum long after the patient has ceased to be exposed to the inhalation of coal-dust, and the physical signs of cavities, together with the hectic fever, emaciation, and sweating, differ in no way from the clinical picture seen in pulmonary tuberculosis, though the bacilli are often not to be detected in the sputum.

In iron-workers the bronchitis and emphysema set in earlier, and in those engaged for a considerable time in such employment they are severe and almost invariably present. The diagnosis is easily made by the history, and iron can be always demonstrated by the blue colour produced by ferrocyanide of potash added to the sputum after treatment with hydrochloric acid. Bronchiectasis follows in due course, and very generally phthisis supervenes. Whether this latter event occurs or not, the clinical picture becomes finally identical with that of pulmonary tuberculosis. In the grindstone and millstone workers and knife-grinders the result is practically the same—the sputum, instead of exhibiting fine particles of

iron under the microscope, shows more or less angular granules of mineral matter, chiefly of silicate of alumina, fine sand, or quartz. In operatives engaged in the manufacture of phosphorus-meal for agricultural purposes from Thomas slag, Ehrhardt found that the highly irritating nature of the dust-laden atmosphere frequently induced an acute and fatal form of pneumonia of the croupous type, the lower lobes being usually affected. The nature of this latter form of pneumonokoniosis is considered to be decidedly more chemical than mechanical.

The prognosis and treatment of anthracosis, siderosis, and chalicosis are those of the chronic bronchitis, interstitial pneumonia, emphysema, and bronchiectasis which exist. The immediate removal of the patient from the deleterious atmosphere must be insisted upon as soon as symptoms of bronchial irritation have declared themselves. The prophylactic treatment is obvious, and consists in the use of such means as will minimise the amount of dust in the air and the employment of respirators to prevent the foreign material gaining access to the lungs.

PNEUMOTHORAX

This is the name given to the condition in which atmospheric air or gas exists in the pleural cavity. Since the phenomenon is almost always accompanied by the exudation of liquid, it is usually more strictly named Hydropneumothorax. When pus exists as well as air, the condition is known as Pyopneumothorax, and when blood is present with the air the term Hæmopneumothorax is used.

Etiology.—An external perforating wound may be the cause, and the condition is produced and may remain for a longer or shorter time when an extensive incision is made by the surgeon in order to rapidly evacuate the purulent contents of the sac in empyema. It rarely follows the puncture made in aspiration, and is seldom produced when the jagged end of a broken rib perforates the pulmonary pleura; it is also a most uncommon event when an empyema or rib-abscess spontaneously discharges itself through the skin.

In a small proportion of cases pneumothorax is produced by perforation of the diaphragm caused by an hepatic or other abscess originating in connection with perforation of a gastric or intestinal ulcer, simple or malignant. Occasionally the condition has been the result of perforation of the œsophagus by malignant disease or injury by a foreign body.

A considerable number of cases are the result of an accumulation of pus in the pleural sac, which ulcerates its way inwards

and discharges into a bronchus, establishing a direct opening between the pleural sac and the respiratory tract.

By far the greater majority of cases of pneumothorax are the result of perforation of the pleural sac from within by diseases originating in the lung. Hence phthisis is the most common cause of the condition by the rupture of a small cavity or caseating focus near to the surface of the lung. It is especially in the more acute forms of pulmonary tuberculosis that the accident occurs, since in the very chronic varieties there are generally extensive adhesions between the visceral and parietal layers of the pleura which prevent the air finding access to the sac.

Gangrene, abscess, pulmonary infarcts, hydatids, and malignant disease of the lung-substance occasionally cause similar perforation. Pulmonary emphysema is not by any means an infrequent cause by the bursting of an emphysematous bleb situated immediately under the visceral pleura in a fit of coughing or during severe strain.

It must not be forgotten that a considerable number of cases of the most typical pneumothorax, in which little or no fluid can be demonstrated in the sac, are found in otherwise healthy individuals, and these are generally associated with the history of some very active recent exertion or physical strain. In one case seen by the writer the condition immediately followed a rapid run up a flight of stairs, the patient falling in a fit of urgent dyspnoea upon the landing. In another the symptoms and signs were not observed till the expiration of forty-eight hours after the individual had been severely mauled in a football scrimmage by a number of players falling over his body as he lay upon the ground. Both these patients made a rapid and steady recovery, and there was no reason to believe that any factor was at work save a rupture of the pulmonary tissue near to the surface of the lung.

Some authorities have attempted to explain the production of gas in a pleural cavity containing serous fluid or pus by the supposition that gas-forming micro-organisms have been at work; others believe that gas may be secreted in the lymph-spaces of the visceral pleura; whilst some authorities attempt to explain the presence of gas or air by the theory that a reduction of the pressure in the sac permits the escape of the gas dissolved under an originally higher pressure in the liquid exudate.

Pneumothorax, however caused, is much more frequently met with in males, and oftenest during early adult life.

Much confusing literature has been created in the attempt to explain by the rigid application of purely physical laws the mechanism of effusion, pneumothorax, and other conditions occurring within the thorax.

Even when a large incision is made into the pleural sac in diseased conditions, as in the treatment of an empyema, the lung, instead of collapsing or maintaining the collapsed condition which was caused by the accumulation of the pus, very generally expands, so that in a few hours both layers of the pleura may be found in close contact with each other. A very wide and patent opening made into the chest-wall, as sometimes happens in accidental wounds received in health, may permit the air to enter and leave the pleural sac without hindrance, in which case the naturally distended lung will certainly shrink or collapse by its own elasticity as soon as the pressure of the air in the sac and the pressure of the air in the lung become reduced to that of the atmosphere. The mediastinum will be also found displaced to the sound side by the elasticity of the opposite lung.

In pneumothorax caused by perforation in pulmonary disease, as phthisis or bronchial ulceration, almost invariably the air from the lung in inspiration reaches the pleural cavity much more readily than it leaves it in expiration. The immediate result is that the pressure of the air in the pleural sac during expiration is considerably greater than that in the lung, so that the latter collapses and great displacement of organs occurs. Displacement of organs may, however, be marked without any increase in the pressure of the air in the pleural sac during expiration.

In the normal pleural sac the pressure is always negative owing to the constant action of the positive elastic pressure of the lung exercised in respiration. In expiration the intra-pleural tension is therefore obviously less than in inspiration. When perforation of the pleura occurs the air in the lung will continue to enter the sac during inspiration till both the pressures become equalised to that of the atmosphere. In pneumothorax, during expiration the pressure is always positive, so that a further and more complete collapse of the lung and greater displacement of organs result than from the mere elastic recoil of the pulmonary tissue.

The intra-pleural pressure is usually much increased by the presence of fluid in pneumothorax.

The opening in the pleural membrane in most cases of pneumothorax is valvular, which explains the fact that the air gains a readier access to than exit from the pleural sac; but as soon as complete pulmonary collapse occurs the opening usually becomes sealed.

Morbid Anatomy.—Upon opening the thorax there may be audible escape of gas owing to its increased pressure. The gaseous fluid is usually of the same composition as the atmospheric air, though the relative proportions of oxygen, nitrogen, and carbonic acid may vary considerably, and sometimes

sulphuretted hydrogen is present. The lung is usually considerably diminished in bulk, often coated with lymph, and sometimes bound down by firm adhesions. The opening in the collapsed lung may be found usually somewhere in the vicinity of the junction of its upper and middle lobes; it will obviously vary much with the primary cause producing the rupture, being sometimes scarcely discernible when an emphysematous patch near the surface has given way. In the majority of cases its connection with a phthisical cavity or with a necrosing caseous deposit will be detected.

The mediastinum is *drawn* over to the sound side and the heart is considerably displaced. As a rule, more or less serous or purulent exudation is present. The nature and amount of this depends upon the severity of the irritant which has reached the pleural surfaces with the air; being obviously greater when a putrid bronchiectasis or old tubercular cavity has discharged its contents than in a simple rupture of the pulmonary tissue from injury or strain or emphysema, where only air has been admitted. The pulmonary tissue appears to be able to filter or sterilise the inhaled air, so that but little irritation follows.

Symptoms.—When perforation of the pleura occurs in chronic phthisis, in which condition there are usually present before the rupture cough, chest-pain, dyspnoea, &c., the primary affection may entirely overshadow the symptoms of the pneumothorax. When air has gained access in healthy subjects to the pleural sac the symptoms are as a rule marked and urgent. Shock or collapse, with the feeling of something having been torn, suddenly sets in. There is an alarming sense of constriction or of distension, with severe agonising pain and dyspnoea, which may rapidly pass into orthopnoea. The anxious face, at first blanched, soon becomes grey or livid, and a cold perspiration breaks out. The pulse is that of collapse—weak, small, and rapid; the extremities are cold. Where a pin-hole perforation has taken place the symptoms will obviously be less urgent, and may only show themselves in a gradually increasing dyspnoea without any evidence of collapse or shock.

Physical Signs.—These are usually so well marked that the condition can scarcely be mistaken for any other.

Inspection.—The posture of the patient is variable; usually he sits propped up, with his *alae nasi* and accessory muscles of respiration in full play and the shoulder of the affected side elevated. The side is obviously distended, the intercostal depressions obliterated, and the respiratory movements greatly diminished or entirely absent. The heart's displacement is great; the writer has seen the apex-beat high up in the right axilla. The movements of the sound side are much exaggerated.

Palpation.—The above signs are all corroborated by placing the hand upon the thoracic walls. Vocal fremitus will be found absent, and the liver or spleen may be felt depressed by the descent of the diaphragm.

Percussion.—The affected side is more or less tympanitic. In rare cases, however, where the tension is high, the note may be almost dull. The hyper-resonance varies from a clear amphoric tone to the flatter sound described as Skodaic resonance. The resonance may be normal over the apex when this is firmly adherent, and dulness is usually marked at the base when fluid has been poured out; this basal dulness shows evident changes with alterations of posture to a degree never observed in ordinary pleurisy with effusion. The cardiac dulness in left-sided cases is usually absent, and when the right sac is distended with air the liver-dulness disappears entirely in the thoracic area.

Percussion by means of two coins may give a characteristic bell-sound, but this is best elicited by a combination of percussion and auscultation. Wintrich's sign may sometimes be observed—*i.e.* the tympanitic sound becomes louder and higher in pitch when percussion is made whilst the patient's mouth is open; and this is supposed to indicate an open condition of the pneumothorax.

Auscultation.—There may be no breath-sound whatever heard in listening over the distended sac when the opening is small or valvular. As a rule, however, a faint distant vesicular murmur may be detected, and this has always some metallic or amphoric qualities added to it. When a large patent opening exists, the air may be heard entering and leaving the pleural chamber, and the amphoric quality of the sounds is striking and the voice and cough sounds are metallic in character. By listening at the back of the chest whilst a coin laid upon the front is struck with another coin or metallic object a clear bell-like tinkle is heard (*bruit d'airain*), the over-tones causing a ringing musical sound.

Metallic tinkling, also known as 'the falling-drop sound,' is very generally present. This sound conveys the idea of being produced by a drop of fluid falling from the apex of the pleural sac into the fluid which has accumulated at its lower part. The tinkle is of an exquisitely metallic quality, and its prolonged after-tone reaches the ear like a distant echo. There is no satisfactory explanation of its production, and the writer has heard it in its most typical form in a case in which he believed no fluid exudate whatever was present.

Hippocratic succussion is a highly characteristic phenomenon, and if dilatation of the stomach or a large phthisical cavity can be eliminated it may be regarded as practically clinching the diagnosis. It is elicited by placing the ear directly in contact with the chest-wall while the patient

jerks his body forwards and backwards or from side to side. Sometimes the splash can be heard by the patient and by those standing at a considerable distance.

A long metallic gurgling sound has been described under the title of 'the lung-fistula sound'; it is audible in inspiration and is supposed to be caused by the air from the lung passing through the fluid exudate in a series of bubbles. Various metallic râles may be audible.

The Röntgen rays may be employed to show the extent of the pulmonary collapse, and the method at the same time may give valuable information about the displacement of organs when the position of the heart cannot be located by inspection, percussion, or auscultation. Also the diagnosis of the primary condition of the lung may be elucidated.

Little attention need be paid to the clinical discrimination between closed and open pneumothorax. In open pneumothorax the wound in the costal or visceral pleura remains patent. In the closed variety the opening in the visceral pleura soon becomes sealed by lymph, after which the air is soon absorbed. In the most common—the valvular condition—the distension of the side and displacement of organs become extreme owing to the increased tension in the pleural sac.

The subsequent course of the disease and the amount of fever and other symptoms present depend upon the nature of the primary malady. In tubercular cases, where a considerable cavity empties its contents into the pleural sac, death may occur in a few hours. The duration and termination of the disease, apart from the influence of the primary malady, are determined by the extent and nature of the exudation resulting from the pleural inflammation.

Diagnosis.—When the majority of the signs already described are present, a mistake in diagnosis is not likely; but where previous adhesions have glued together portions of the opposite pleural surfaces, the signs may be confusing if pneumothorax supervenes upon such a condition. The resulting encapsuled pneumothorax may then closely simulate a large *phthisical cavity*. In the latter condition, however, there must be obviously a clear history of long-standing failure of health and of the symptoms which always accompany a chronic and extensive pulmonary cavern. As pneumothorax, however, is so frequently found to follow phthisis the encapsuled form may be present as the result of a small caseating focus rupturing in an emaciated patient. The following points will distinguish it from a large phthisical cavity. The most important of these is the displacement of the heart, which is always away from the air-containing pleural sac; whilst in phthisical cavity the heart is dragged toward the diseased lung. The discovery of tubercle bacilli

in the sputum argues in favour of a cavity, but they may be coming from a small focus at the apex of the lung, whilst a pneumothorax may exist from a perforation lower down. It is extremely rare for the bell-sound and Hippocratic succussion to be heard over phthisical cavities, but such phenomena, when they occur, may be associated also with metallic tinkling and amphoric breathing. A point of great diagnostic value in such a case is the marked retraction of the side, which contrasts with the distended condition of the affected half of the thorax in pneumothorax, with its very extensive area of tympanitic resonance and obliteration of the intercostal spaces.

Pulmonary emphysema need not be thought of as a difficulty, except in those rare cases where dyspnœa suddenly supervenes owing to a rapid failure of cardiac compensation, followed by acute dilatation of the right ventricle. The chest-walls give out a resonant note, and the area of cardiac dulness may be entirely obliterated. The condition is, however, always bilateral, and there is no displacement of organs; a bell-sound cannot be elicited, and splashing phenomena are absent.

Two points in the diagnosis of pneumothorax must be constantly remembered: the first is, that as it is nearly always the result of some primary lung-disease, the aim of the physician should be directed to the diagnosis of the causative factor; and secondly, that in all doubtful cases the final pleural inflammation and effusion at the bottom of the sac should be investigated both with the view to diagnosis, prognosis, and treatment.

Puncture of the air-containing sac does not always give evidence of high intra-pleural tension; even when much displacement of the heart is present the air may not escape through the needle unless dyspnœa be marked or unless considerable liquid effusion has taken place.

Subphrenic pneumothorax is the condition in which an air-containing abscess-cavity exists beneath the diaphragm, the result of perforation of the stomach, colon, appendix, gall-bladder, &c., or of any condition causing peritonitis. The diagnosis of such a condition from pneumothorax will be easy when the history of the case and its abdominal origin are clear. The intercostal spaces are not distended, the heart is usually little, if at all, displaced, and true vesicular breathing is audible over the affected side of the thorax except at its lowest part.

Diaphragmatic hernia is recognised by its distinct abdominal symptoms—meteorism, persistent vomiting, and intermittent colicky pain, with obstruction of the bowel, &c.

Pleurisy with effusion may be simulated by a pneumothorax seen at a late stage, when much fluid exudate has been

poured out and a considerable amount of the gas has been absorbed by the pleura. The presence of Hippocratic succussion with the bell-sound phenomenon will settle the diagnosis in such a case, and prevent the tympanitic percussion-sound below the clavicle being mistaken for Skodaic resonance. Moreover, the remarkable mobility of the dulness in pneumothorax upon changes of posture is almost characteristic.

Prognosis.—The gravity of the outlook depends largely upon the nature of the primary cause. It is always most grave when occurring in the advanced stages of phthisis, and death may result in a few hours or days, and rarely is the fatal result protracted beyond a few months in such cases. Left-sided pneumothorax under such conditions is somewhat less serious than when the perforation is on the right. The nature of the resulting exudate is an important element in the prognosis, being much more serious in purulent cases. An important fact must not be forgotten, that in early phthisis the supervention of pneumothorax exercises a distinctly inhibitory influence over the progress of the tubercular process in the lung upon the affected side; and numerous cases are upon record where complete recovery has followed, even when the tubercle bacillus had been detected in the sputum. This fact has encouraged Forlanini to treat early phthisis by the artificial production of pneumothorax and his results, recently reported (*'Deut. Med. Woch.'* August 1905), are most interesting. He gives details of this treatment in twenty-five cases, the majority of which were satisfactory when the disease was limited to one lung. The remarkable fact is proved by his method that the patients lead active useful lives with voluminous pneumothoraces. The essential point in his treatment is the pumping of nitrogen gradually into the pleural sac so as to cause a voluminous pneumothorax in order to completely put the affected lung out of action and ensure its absolute rest by destroying the mobility of the thorax on the side where the tubercular lesion exists.

The prognosis in simple traumatic pneumothorax caused by rupture of the lung in healthy individuals is very favourable. The writer has witnessed perfect recovery follow in two cases. Even in empyematous cases the formation of a permanent fistula is compatible with prolonged life under comparatively comfortable conditions.

Treatment.—The symptoms of shock, the pain, and dyspnœa are to be met by morphia hypodermically and by stimulation upon recognised principles. Unless there be unmistakable evidence of greatly increasing intrapleural tension, it will be advisable simply to keep the patient at rest and treat symptoms as they arise. A fine trocar may be thrust between the ribs so as to permit of the escape of the imprisoned air when the presence of a valvular opening

causes such a degree of cyanosis and asphyxia as threatens life. The operation should, however, only be undertaken when the condition is unmistakably urgent, as the aim of the physician should be to encourage nature to seal up the opening in the collapsed lung.

Aspiration should never be resorted to, the air being permitted to force its own way out through the cannula. Strapping of the side may be advantageously resorted to after the evacuation of the air, but without puncture such a procedure can only embarrass the increased compensatory action of the sound lung.

At a later stage the nature of the fluid poured out should be investigated by needle-aspiration, and if found to be purulent a free incision should be made and the case treated upon the principles recognised as suitable in empyema. Serous effusions are best left alone: little good and much harm may result from frequent aspirations. In advanced phthisis obviously the presence of pus may not justify a free incision, the operation being contra-indicated by the grave general condition of the patient. Sometimes life may be saved, however, in case of pneumothorax caused by the rupture of a gangrenous cavity in the lung or by an empyema, even when the constitutional symptoms are of the gravest type.

In traumatic cases, where an external wound has permitted the entrance of air into the pleural sac, septic organisms are certain to be present; as a rule, the opening should be freely enlarged and irrigation effected upon general surgical principles; free drainage should be also established.

POLIOMYELITIS.

(See under PARALYSIS, INFANTILE, p. 1193.)

POMPHOLYX

This is the name given to an uncommon disease characterised by a bullous and vesicular eruption appearing upon the hands and feet. It is also known as Dysidrosis, and owing to the hands being most frequently the site of its manifestation it was named by Hutchinson Cheiro-pompholyx.

Etiology.—It occurs in young subjects, but is not met with till after puberty. Debilitated women of neurotic temperament constitute the majority of the patients; it is not seen in aged subjects. It is most frequently observed in patients whose hands and feet are liable to sweat.

Symptoms.—The eruption usually makes its appearance in hot weather upon the hands, preceded or accompanied by

a burning or tingling sensation. The vesicles are deeply situated beneath the horny layer of the skin along the sides of the fingers and palms. Groups of vesicles coalesce into bullæ of irregular shape which project above the level of the skin ; the contents are at first clear and pellucid as in herpes, and there is little redness. The conglomeration of vesicles may reach to the size of a shilling ; upon puncture a watery, albuminous, neutral or alkaline fluid is obtained, which in the older vesicles or bullæ may be ropy or turbid. In severe cases the entire hand (dorsal and palmar aspects) is affected ; often the feet participate, but sometimes they escape entirely. The eruption is always bilateral. After about ten days the bullæ dry up without spontaneous discharge, and desquamation follows, leaving a layer of new rosy, healthy, dry skin. Recurrence is the rule, another attack following later in the same summer or in the following year.

Pathology.—The pathogeny of the affection is doubtful ; it is believed by Unna to be the result of a specific bacillus. Others maintain the view first promulgated by Fox, who considered that the diseased process started in the sweat-glands either as a retention of their secretion (dysidrosis) or as a hyperidrosis. The anatomical changes are variously described by different observers, the most constant being those indicative of a mild inflammatory process in the papillary layer, which is infiltrated by leucocytes in the neighbourhood of the vessels supplying the papillæ.

Diagnosis.—The natural tendency of the affection towards spontaneous resolution and recurrence, its site, and the fact that the bullæ are formed by coalescence of vesicles and that no weeping follows desquamation, distinguish it from *eczema* and *pemphigus* and all other eruptions which appear upon the hands or feet.

Treatment.—Simple ointment of zinc or any other bland astringent application is all that is necessary to relieve the tingling of this short-lived affection. Recurrences are prevented by tonic treatment and improved hygiene.

PRURIGO

This is a cutaneous disease accompanied by intense pruritus and characterised by the presence of a papular eruption, usually upon the extensor surfaces of the limbs and both aspects of the trunk.

Etiology.—The disease is usually confined to ill-fed and badly clad poor subjects, in whom it always first appears in early childhood or infancy, and remains commonly all through life subject to remissions and exacerbations. Males are much more commonly affected than females. In some patients

the disease appears to be greatly aggravated by hot and in others by cold weather, but in all cases it is probably intensified by sudden variations of temperature.

Symptoms.—Two forms of the malady are recognised ; one—*prurigo ferox*—is very uncommon ; in it the itching is most intolerable and the eruption extensive and accompanied by severe secondary lesions, with obvious changes in the subcutaneous tissues. The second form—*prurigo mitis*—is the one most frequently met with and to which the following description applies.

The papules are of the size of about an ordinary pin's head or hemp-seed, and at first are of the colour of the skin ; they are never grouped or found in coalesced patches, but remain isolated and scattered throughout. The disease appears usually to commence, according to Crocker and Comby, as a lichen urticatus or a papulose urticaria, being at first commonly associated with large and small wheals during infancy. These latter disappear later on, and after a few years the papules often alone remain when the prurigo has assumed its typical development. The papules exist in greatest number upon the extensor surface of the lower extremity below the knee and outer surface of the thigh, upon the back of the forearms, the front and back of the chest, and the lower zone of the trunk, sacral region, buttocks, and dorsal aspect of the feet. Very rarely, if ever, are the soles, palms, arm-pits, wrists, or ankles involved, and the flexures of the knee and elbow and the genitals always escape. Though a few papules often appear upon the face, the scalp and neck do not suffer.

The chief clinical symptom is the intensity of the itching or pruritus, which causes severe scratching, so that the eruption, which is at first only to be felt by passing the finger-tips over the pale papules, soon becomes very obvious as the pallor gives way to a deep reddening of each little elevation, which is usually topped at its summit by a small blood-crust or scab.

The health suffers little unless the temperament of the patient be such as causes serious insomnia from the itching.

Severe cases of *P. mitis* differ in no way from *P. ferox* or Hebra's Prurigo save in intensity, and there is no line of demarcation between them. In a well-marked case of the graver type the following signs may be observed, all of which are present during exacerbations in the minor form in a less degree—viz. more or less traumatic eczema from scratching, accompanied by thickening of the skin and deepening of its natural furrows ; the subcutaneous fat is diminished. The sensation conveyed to the hand of the physician upon examining the affected skin has been likened to that experienced by passing the fingers over hard, coarse, brown paper or

over the surface of a nutmeg-grater. Crusts and scabs are frequently seen forming upon the wounds made by the fingernails. The neighbouring lymphatic glands become indurated, large wheals appear after scratching, and finally there is considerable increase in the skin-pigmentation, and the fine hairs, if not destroyed in the scratching process, stand out stiffly in the affected areas. The lichenification of the skin is seen in its hypertrophic thickening and in the mealy exfoliation which is always occurring in old cases.

Pathology.—The pathogeny of the affection is doubtful, but most authorities regard it as a neurosis, though Hebra maintained that the pruritus was always secondary to the primary papules in the graver form of the disease. The papular eruption, skin induration, and other changes are regarded by many as merely secondary results following the scratching, which itself is caused by some toxic agent circulating in the blood and irritating the peripheries of the nerves supplying the skin.

Diagnosis.—*Vagabond's disease* (*phthiriasis* or *prurigo senilis*) closely simulates the condition, but the discovery of pediculi and the history of the case settle the diagnosis easily.

Chronic eczema is also eliminated by the history of the case, which, as already stated, always dates from infancy or early childhood; the characteristic site in which invariably the flexor surfaces are always spared is an important point in the exclusion of eczema and other affections.

Lichen urticatus, or simple prurigo or urticaria papulosa, as it is also called—an affection common in infancy in the summer months—cannot be excluded till after the expiration of a considerable time, and sometimes the differential diagnosis is impossible. The site of the flattened papules in this latter disease is not so circumscribed as in prurigo; though the flexures may escape, the soles and palms, the scalp and face, often suffer. Vesicles commonly replace the papules as the itch intensifies, sometimes simulating varicella. An important diagnostic point, as emphasised by Fox, is the frequent presence of erythematous macules centred by the papules. There is no tendency to enlargement of the neighbouring lymphatic glands, and lichenification of the skin does not occur in simple prurigo, though a secondary eczema may result sometimes and an ecthymatous condition may be superadded. The importance of the subject lies in the fact that many observers have traced the gradual progress of this simple prurigo of infancy into Hebra's grave variety, and also because the only hope of cure in prurigo exists in early and persevering treatment.

Prognosis.—This is always serious as regards the prospect of a complete cure, though in mild cases the disease can be alleviated very considerably by appropriate symptomatic

treatment. In cases seen early the prospects are brighter. In *P. ferox* the prognosis is always bad.

Treatment.—The chief indication is for remedies which relieve itching and which minimise, remove, or prevent the secondary lesions. At the same time, every agent which improves the general nutrition and raises the standard of health must be perseveringly employed.

Of local remedies, warm baths containing weak alkalies, starch, carbolic acid, creolin, &c., are useful; they allay itching and may advantageously be followed, immediately after drying of the skin, by inunctions of lanolin, vaseline, or cod-liver oil, combined with tar, carbolic acid, menthol, naphthol, or oil of cade. Massage may be advantageously employed in conjunction with the above applications. When severe eczema, impetigo, or ecthyma complicates the condition, mild emollients should be employed at first. Of internal agents, although none are known which exert a specific action, cod-liver oil is decidedly beneficial. Kaposi recommended carbolic acid, and Crocker cannabis indica; thyroid feeding and antipyrin have their advocates. Iron, arsenic, phosphorus, and other alteratives may be tried from time to time in courses of short duration. Pilocarpine and cocaine hypodermically may be indicated as temporary measures when the pruritus is extreme, in which case the patient should be confined to bed and his sleep assisted by the administration of hypnotics; but opiates are, as a rule, contra-indicated in this condition. The writer has seen considerable relief follow the administration of chloride of calcium when the urticarial signs were prominently marked.

PRURITUS

This is defined clearly by Morris as 'itching without any visible cause to account for it,' and if the definition be kept in mind all the confusion existing about pruritus and prurigo disappears. Many dermatologists, however, employ the term loosely as a synonym for itching; thus, as in the previous article, the itching which is the chief clinical feature in prurigo is termed pruritus; this is, however, from a pathological point of view, justifiable, since, as already stated, many authorities, chiefly of the French school, regard prurigo as a neurotic affection, the eruption itself being, according to their view, but the result of the scratching. The pathology of pruritus is that of a sensory neurosis, peripheral or centric.

More or less itching is characteristic of a host of skin-affections, with definite and unmistakable anatomical lesions, as eczema, scabies, pruritus senilis, urticaria, &c.

The term 'pruritus' in the present article being accepted, according to the above definition, as itching occurring in an apparently normal skin, the affection may be regarded as divisible into two distinct clinical groups—*general* and *local* pruritus.

General pruritus is seen in its most typical variety (*P. universalis*) in conditions like jaundice, where the itching sensation is experienced over the entire cutaneous surface, one portion of the skin after another being at different times affected by the sensory irritation. Gout, kidney-disease, diabetes, stomach and uterine affections, pregnancy, rheumatism, and lithæmia produce a similar condition. If the symptoms of the primary affection be omitted, practically nothing remains but the itching and the secondary traumatic lesions, insomnia, &c., which follow. The affection usually is more marked in cold weather, but is always aggravated by the heat of the bed-clothes, being almost invariably worst at night.

In the variety known as *P. hiemalis*, occurring chiefly in rheumatic subjects, the itching is confined to the winter season and only becomes as a rule marked at night. In *P. senilis* pediculi are nearly always the exciting cause, but some dermatologists recognise a form of this affection which they consider to be the result of senile changes in the skin which may exist independent of parasites.

It must not be forgotten that a mental condition may be the chief or only factor in producing a general pruritus. This variety of the affection in its most common form is usually transitory. It may be seen in sensitive subjects who have accidentally discovered pediculi upon their persons or clothing or in their immediate vicinity, after which severe pruritus is experienced in different regions of the body for many hours or days, the itching being apparently prolonged by the scratching. That the discovery of the accidental presence of the parasite is not coincident with some blood or other condition is obvious, since the attack soon passes off and never returns, unless under similar circumstances. The mental influence is probably a potent factor in the aggravation of pruritus, by whatever cause induced. This is the obvious explanation of the variety recognised by Crocker under the name of *P. mentis*, which sometimes eventuates in melancholia.

Local varieties of pruritus affect the anus, vulva, scrotum, and sometimes the palms and soles. In the great majority of cases there is a local cause; thus pruritus of the anus or perinæum is very often produced by the presence of a small fissure, hæmorrhoids, thread-worms, and scybalæ, but the cause in some instances may be gout, hepatic disease, or diabetes.

Pruritus vulvæ is very often the first symptom of diabetes in women, being either the result of the local irritation caused

by trickling of the saccharine urine over the sensitive area where the skin and mucous membrane join, or else being a local manifestation of the constitutional condition. In either case the condition is associated always with more or less eczema. Irritating vaginal discharge, ovarian, uterine, or other pelvic tumours or diseases, are liable to set up the disorder, which may, however, also appear as a local manifestation of the neurotic condition often associated with the cessation of the menses at the climacteric period.

In some cases, especially about the genitals, the pruritus is the result of hyperidrosis, and is then frequently associated with intertrigo.

The scrotal form is very rarely seen, unless when caused by eczema or intertrigo, in which case it cannot be strictly regarded as a pruritus. The head of the penis may be the seat of severe itching, caused by the direct irritation of a slight urethral discharge or stricture, which is liable to be overlooked; in other cases, especially in children, it may be a reflex from a vesical calculus which produces no other symptom.

The pruritus of the nose, popularly considered as an unfailing symptom of ascarides, is probably the result of intestinal catarrh.

Pruritus of the soles and palms in its typical form is a rare affection seen usually in gouty subjects and in women sometimes at the climacteric period; the 'itching palm' is usually found associated with chronic eczema and much thickening of the epidermis (eczema rimosum).

Diagnosis.—The first step in the recognition of the disease is the exclusion of all affections caused by parasites. Scabies and pediculi are the cause of the great majority of all conditions liable to be mistaken for the sensory neurosis constituting true pruritus. In this consideration the chief point in the differential diagnosis is the site of the injuries caused by the finger-nails. These in *phthiriasis* are usually about the shoulders, whilst in *scabies* the spaces between the digits, the wrists, and the inside of the thighs are the common sites, the parasites or their burrows being evident.

Urticaria can never be mistaken for pruritus when the eruption of raised patches or wheals is visible. In the more chronic variety, where the eruption only appears at night, the condition may be mistaken for pruritus, but the history of the case reveals its true nature. In most severe cases of urticaria at any time mere scratching, when there is no sensation of itching, will bring out a crop of wheals.

The papules of *urticaria papulosa* will be always found when looked for, especially when pressure is applied to the skin, and the disease is chiefly confined to children.

Eczema and every other skin-affection is to be eliminated by the absence of their characteristic anatomical lesions and

a careful investigation will always prevent the traumatic scratch-marks seen in such conditions being mistaken for those occasionally produced in true pruritus, since the skin between and around the injuries is always normal.

The condition being determined by the above process of exclusion, the diagnosis of the causes already mentioned must be undertaken by a routine examination of the urine and different organs and a scrutiny of the patient's history. In the local varieties of the disease the diagnosis is established with the discovery of the cause, as an anal fissure, hæmorrhoids, vaginal discharge, or uterine disease.

Prognosis.—This will depend upon the amount of hope which can be fairly held out regarding the removal of the primary cause. Diabetic patients may, however, be often promptly relieved of all pruritus by the administration of a rigid diet, though the primary disease is only alleviated for the time. The prognosis in glycosuric, rheumatic, and gouty patients is also hopeful, and the local varieties of the disease as a rule yield speedily upon the removal of the primary local cause.

Treatment.—The chief indication obviously is for the removal of the causative factor when this is possible. But even in a condition like jaundice the pruritus may be considerably relieved, when the obstruction is irremediable, by the internal administration of pilocarpine or of gelsemium, atropine, tartar emetic, cannabis indica, or the newer analgesics; the first-mentioned drug should be given hypodermically. Elimination of gouty and other products through the bowel and kidneys by saline purgatives and diuretics is clearly indicated in most cases, and hence success often follows a course of alkaline mineral-water at a suitable spa. The diet must be closely scrutinised, not only with the view of the treatment of the diabetic, gouty, or rheumatic diathesis, but many patients suffering independently of these diseases may find at times the pruritus greatly aggravated by some article of diet harmless to other persons. Massage and electricity in the form of faradism or galvanism, or in the static form, or as high-frequency currents, have all at times proved valuable in both the general and local varieties of pruritus.

Local treatment is indicated in all cases; thus in the general or universal variety of the disease warm alkaline baths are very serviceable.

Applications innumerable are vaunted for the relief of local pruritus, but without a removal of the local source of irritation little permanent benefit is to be expected. For anal pruritus the writer finds that nothing is to be relied upon except the ointment of conium, which in many cases seems to exercise a specific action. Crocker extols mercurial ointments. Cocaine and menthol, oil of peppermint, chloral, tar, chloroform,

carbolic acid, camphor, and other local anæsthetics, whilst often giving transient relief, generally, in the long run, tend to aggravate the malady. Pruritus vulvæ is best treated, after the vaginal, uterine, or pelvic cause is removed, by alkaline sitz-baths, by warm borax or lead lotions, or by the unguentum conii. Relief may sometimes be obtained by free dusting of the mucous surfaces with zinc oxide, bismuth carbonate, or other soothing impalpable powder, especially if excoriations have been produced by scratching.

The rarer palmar and plantar varieties can only be treated by constitutional medication directed towards the gouty, rheumatic, or climacteric states, which have induced more or less dry eczema, with induration of the skin.

PSORIASIS

This is a common cutaneous affection, generally of very chronic duration, characterised by the presence of flat, circumscribed, round, or gyrate patches covered with silvery scales.

Etiology.—The condition may appear at any age, though rare after sixty and in early childhood; in the majority of all cases the disease first shows itself before puberty. Sex seems to have no etiological signification, neither has social rank, occupation, nor season, though this latter may be found to be a factor in the intensification of the established condition, many cases being aggravated in the spring and autumn months. It is more common in northern latitudes than in hot climates, and the negro race is much less susceptible than whites.

Heredity plays some part, a family-history of the disease being obtainable in from 10 to 15 per cent. of cases.

It has been noted as following in some instances upon an abrasion or injury of the skin, and occasionally it has been observed to supervene after vaccination and erysipelas.

The disease cannot, in the ordinary sense, be regarded as contagious, but unquestionable examples, though exceedingly few in number, have been observed in which the affection was conveyed by contact; and Destot succeeded in producing the disease by experimental inoculation in his own person. No specific parasitic organism has yet been isolated. Violent mental emotions and psychical disturbances have preceded the appearance of the affection sufficiently often to establish the fact that such conditions are capable of at least acting as exciting causes.

The theory that gout, rheumatoid arthritis, hyperlactation, and other depressing conditions act as etiological factors rests upon no solid foundation. In the majority of cases the

disease appears in the most typically healthy subjects, so that practically nothing is definitely known of the etiology of the affection, save that heredity plays some feeble rôle in its incidence.

Symptoms.—The eruption first appears as a flat, dull, red papule, often not larger than a pin's head, which, within a few days or from its earliest commencement, is covered with white silvery scales. It gradually enlarges circumferentially, and rises somewhat above the level of the healthy skin, from which it is sharply defined by its definite border, so that an irregularly shaped or rounded patch finally results, of considerable dimensions. A large patch or plaque, however, may usually be observed to develop from the coalescence of several smaller ones, situated closely together, and such a patch may remain stationary for months or years. The scales are large and adherent, and present a distinct newly minted silver or mother of pearl lustre; they form a layer of variable thickness over the patch, and when scraped off by the finger-nail a hyperæmic base, showing a number of fine red points, is visible. These are the tops of the chronically inflamed or hyperæmic papillæ, and further scratching or scraping will cause them to readily bleed. Psoriasis is essentially a 'dry' disease, so that no exudation, weeping, or crusts are produced as in eczema, impetigo, &c., and as a rule there is very little or no itching.

The usual site of the eruption is the extensor aspects of the limbs, and the physician always looks for it upon the front of the knees or back of the elbows; if found here, there is very often some traces of it to be found also on the hairy scalp. The trunk is also more or less involved in most cases, but the disease may for a long time only show itself by the patches upon the knees and elbows. There is always more or less of a distinct symmetrical character in its distribution. This may not be obvious when the patches are confined to the trunk, but one knee or one elbow is practically never seen to be alone affected, and the same remark applies to the palms and soles, which very occasionally also suffer.

An infinite number of names have been coined to designate the varieties of the affection. These are of little importance, however, since in all cases the diseased process is the same, the variations in the appearance of the eruption being mainly the result of the isolation or coalescence of the patches. Thus, when the primary scale-covered papules remain discrete, and do not increase in size, the term *P. punctata* is applied; when the discrete plaques are somewhat larger they resemble small splashes of mortar, and this variety is designated *P. guttata*; *P. nummularis* is the name applied to the round coin- or disc-shaped patches; when the patches coalesce to

form large irregular areas of eruption the condition is known as *P. diffusa*; and when these remain for a long time without resolving there is much thickening and sometimes fissuration—a condition to which the term *P. inveterata* is applied.

The patches ultimately tend towards involution or resolution, which commences in the centre; this becomes pale, and loses its scales, so that the disc- or coin-shaped area becomes transformed into a ring—*P. circinata*. In a rare variety known as *P. rupioides*, conical concentric masses of scales are piled up, limpet-shaped fashion, as in *rupia*.

As the resolution proceeds in one part of the patch the diseased process may be extending in another, so that a condition results to which the name *P. gyrata* is applied, the remnants of one ring uniting with those of others, forming festoons of varied appearance. The old term *Lepra* was applied to those cases where the rings remained unbroken but continued to spread circumferentially whilst resolving in the centre, the width of the diseased or spreading ribbon of the eruption not increasing.

As final resolution occurs the skin becomes normal in every respect, save that often some pigmentation remains, even in cases where no arsenic had been administered; very occasionally only has the resolved skin been observed to remain bleached, or leucodermic, or scarred. In a few reported cases keloid, epitheliomatous, and verrucose growths have been observed to develop upon the site of an old patch of psoriasis. Where the eruption has been subjected to the friction of the clothing, fissures occasionally result which cause considerable irritation; these are liable to form when the scrotum is attacked, and a thin ichor may trickle from the cracks, but a secondary eczema or impetigo is very rarely observed.

When the scalp is affected the scales may be exceedingly abundant, sometimes forming knobs of piled-up brownish epithelium. The growth of the hair is, however, little interfered with, and baldness never occurs, though in severe cases the hair may be thinned considerably. The diseased process in the scalp often ends abruptly with a hyperæmic border above the forehead close to the hair.

Upon the palms and soles psoriasis is rare; when the disease does attack these regions there is much thickening of the horny layer, and the nails are affected often when the palms are free. The disease in the nails shows itself in two forms. In one variety it begins in the matrix: the nail is lifted up by the accumulation of the scales, it splits, and is finally shed. In the other form, the disease, as described by Hutchinson, shows itself in a discoloration, commencing under the free margin of the nail, which becomes thickened as the discoloration gradually advances downwards towards the lunula.

Crocker maintains that the majority of the cases recognised as palmar or plantar psoriasis are examples of palmar or plantar eczema, or else are the result of syphilis.

As already stated, the disease is usually a very chronic one, but it is liable to acute exacerbations; thus, under some depressing condition—mental, physical, or traumatic—a patient who has had old indolent patches confined to the knees or elbows may suddenly find a copious punctate, guttate, or nummular eruption appear over the greater part of the trunk.

In a few cases the disease appears in the first instance as a distinctly acute affection—*P. acuta*—in which, without the production of much scales, bright-red patches come out with indefinite margins over a considerable area of the trunk and extremities, accompanied by severe itching and hyperæsthesia. This form may simulate eczema closely if scratching be freely indulged in, but after a short time the development of the silvery scales demonstrates the nature of the case.

Recurrence is the rule; rarely does a patient ever remain quite free from subsequent attacks, even after complete resolution has occurred.

It must also be remembered that the disease in its acute form, and sometimes also in its most chronic type, exhibits a tendency in some cases to pass into or be transformed into pityriasis rubra, constituting the secondary form of exfoliative dermatitis.

Pathology.—Many theories have been invented to explain the nature of the disease and to interpret the anatomical lesions observed. These latter in the main consist of great hypertrophy of the horny and papillary layers of the skin, with extensive growth downwards of the interpapillary cones. The papillæ are increased in size, and the changes indicative of inflammation are observable in the upper part of the corium in the neighbourhood of the sweat-ducts and hair-follicles. The lustrous appearance of the scales is seen to be due to the presence of minute quantities of air within them, and between contiguous cells.

The diseased process is maintained by some to be of a purely inflammatory nature originating in the papillary layer, whilst others hold to the view that the entire changes are due to a keratolysis, parakeratosis, or anomaly of the cornification-process. Some authorities regard the process as originating in an epithelium hyperplasia of the elements in the rete, to which the inflammatory action in the corium is a secondary event. Crocker believes that the above changes are primarily due to the presence 'of a microparasite, which is probably very widely spread, but only grows in certain persons, and that heredity is really tissue-suitability for the growth of the organism.' This theory fits in with the clinical features of the disease, and explains the facts regarding its occasional trans-

mission and inoculability better than does the older view of its pathogeny—viz. that it was due to a primary neurosis of the skin of tropho-neurotic or vaso-motor nature.

Diagnosis.—The disease, when seen in its typical condition, uninfluenced by treatment, can scarcely be mistaken for anything else. The symmetrically distributed patches, with their imbricated silvery scales situated upon the front of the knees and back of the elbows, with the sharp definition of their borders, the red bleeding-points visible in the hyperæmic base when the scales are scraped off, and the generally robust health of the patient all form features in a clinical picture quite unmistakable, especially when it is recognised that the affected areas of skin are throughout devoid of any secretion or weeping exudation.

Eczema is eliminated by the last-mentioned feature of dryness, another differential point being the almost entire absence of itching in psoriasis. In eczema the favourite situation is upon the flexor surfaces; its very beginning is in a group of vesicles, differing entirely from the initial dry flat papules of psoriasis. Even when there is little secretion the crusts of eczema, formed out of the dried-up serous discharge, reveal the nature of the case. The eczematous patch is never sharply defined from the surrounding healthy skin. In the old dry eczemas the crusts are replaced by scales, but these never present the imbricated and shining characters of the circumscribed mortar-splashes of psoriasis. The skin for a considerable distance beyond the scaly patch of an old eczema is found to be thickened and coarse, and nearly always a history of previous oozing may be obtained; usually upon close examination a few small crusts may be found which, when forcibly removed, always reveal, after the lapse of a short period, traces of a fluid serous exudate.

The ruddy glow of health in the subject of psoriasis is generally in marked contrast to the pasty complexion of the eczematous patient, who is obviously below the healthy standard.

Only in dry *seborrhœic eczema of the scalp* is the differential diagnosis usually difficult, but the abrupt termination of scalp-psoriasis above the forehead near to the hair is characteristic, and there are no eczematous patches behind the ears or upon the face, as usually observed in the downward-spreading tendency of seborrhœic eczema, which invades the entire scalp and does not appear in patches. In all doubtful cases the front of the knees and elbows and the finger-nails should be carefully examined.

Pityriasis rubra is always universally distributed and of very rapid onset, often involving the entire surface within a week or two; its thin, loose, papery, or parchment-like transparent scales are characteristic.

Lichen planus selects the flexor aspects. The disease commences in flat, waxy-looking, glistening, smooth papules; scales, if present, are always scanty; the individual papule does not enlarge as does the initial papule of psoriasis, but the lichenous patch is formed always by an aggregation or coalescence of many papules. In scratching away the scales of lichen the basis of the patch is of a bluish or pale purple tint, contrasting with the red base of psoriasis when similarly examined. In very old patches of lichen the surface is horny and raised, the skin much infiltrated, and the itching of a severer form than that ever met with in psoriasis, and the pigmentation or staining is usually more obvious.

Syphilitic skin-eruptions, both in the secondary and tertiary stages, may so closely simulate psoriasis that a diagnosis by inspection alone may be impossible. The site of the eruption seldom confines itself to the extensor surfaces. It has a marked tendency to form gyrate figures; the bleeding-points observed after scraping away the scales in psoriasis are not seen. The face is often involved, and usually other forms of eruption may be observed upon different parts of the body, the tendency of syphilitic eruptions towards polymorphism being always marked. The papular syphilitic eruption appearing upon the palms and soles in the secondary stage, being always symmetrical, is more likely to be mistaken for psoriasis than the similar tertiary eruption, which is often unilateral. In all cases the history will usually settle the diagnosis, when taken in conjunction with the evidences of the disease obtained by an examination of the visible mucous surfaces, eyes, lymphatic glands, testes, bones, &c.

Lupus erythematosus is differentiated by its selection of the cheeks, which generally escape in psoriasis. The edges of the patch are raised, reddened, and infiltrated; there is some scarring at places, with atrophic changes, and the horny plugs which lodge in the dilated mouths of the sebaceous ducts are highly characteristic. The history in the case of *lupus vulgaris* is always distinctive, and the discovery of the characteristic apple-jelly nodules, which do not lose their characteristic colour when pressure is applied, affords a pathognomonic sign of that disease.

Prognosis.—This is good in nearly all cases as regards the existing attack yielding to more or less persevering treatment, but in the great majority of cases recurrences at longer or shorter intervals are the rule.

Treatment.—There is seldom any indication for the administration of drugs with the view to improve the general standards of health and nutrition, since these are usually apparently highly satisfactory. From an empiric point of view certain agents are known which, when administered internally, appear in many cases to exert more or less of a

specific action on the disease, whilst in other cases they fail entirely, or may even aggravate the malady. It is recognised that the best results are obtainable by a combination of internal and external treatments, with due attention to every hygienic measure which secures freedom from skin-irritation, as soft clothing, life in an equable temperature, &c.

Arsenic is the most reliable of all drugs. Its best effects are observable in the most chronic cases; often it fails or causes aggravation of the disease in its acute form. It should be pushed till the physiological effects of the drug become manifest in a mild degree, after which the dose should be diminished. There is no clear evidence of the manner in which its effects are produced. After prolonged courses, hyperkeratosis of the soles and palms has been observed, and very occasionally peripheral neuritis has been produced. The writer has, in some instances, observed excellent results from arsenic in the form of cacodylate of sodium, but the difficulty of prolonged treatment by this drug is greater than when Fowler's solution is administered. The latter can often be safely kept up continuously till after the disappearance of the eruption.

Iodides in colossal doses have sometimes given excellent results, 1 dr. of sodium iodide three or four times daily having been often administered. The drug may also be combined with arsenical preparations. Thyroid feeding, turpentine, tartar emetic, phosphorus, carbolic and salicylic acids, chrysarobin, copaiba balsam, and mercury all have been found beneficial in some cases; they constitute but a short list of the internal agents vaunted as specifics from time to time.

Local treatment is accepted by common consent as of more importance than internal medication, though the numerous hosts of formulæ employed by different dermatologists demonstrate the fact that as yet no specific application is known. In the author's 'Dictionary of Treatment' (4th ed.), pp. 806-813, a list of these is given, and only the recognised principles of the local treatment need be here mentioned. The local treatment consists, in the first instance, of the employment of such measures as effect the removal of the scales. Warm baths containing weak alkalies, as bicarbonate of soda or borax, are useful for producing softening and detachability of the crusts after prolonged immersion. Soap, salicylic acid, or other solvents may be employed afterwards with friction to complete the removal.

In the acute and sub-acute forms of the disease friction is inadmissible; the patient may be permitted to remain for half to one hour or more in a warm starch or bran bath, after which gentle drying of the surface only is permitted. Any bland animal fat or oil may then be lightly applied with a soft

cloth, to saturate the scales and cause their detachment. Old localised patches may be deprived of their scales by warm compresses, continuously applied under oiled silk or other impervious dressing. The scales can be removed from the scalp by using borax instead of ordinary soap.

After the removal of all scales and crusts by persevering and judicious management, the next step in the treatment, as soon as the resulting irritation has subsided, is the application of a weak tar ointment. The best routine tar application is oil of cade, starting with a 1 in 4 ointment, gradually increasing in strength till the pure oil is tolerated. It is often employed in combination with soft soap and sulphur in the chronic cases. The B.P. ointment of wood tar, or an ointment of coal-tar solution (1 in 4) may be applied freely to inveterate patches.

Old circumscribed patches yield rapidly to a chrysarobin treatment; the drug may be ground into a smooth creamy paste, with water or spirit, and brushed over the patch after removal of the scales. As soon as drying is complete the paste is covered by a larger-sized area of adhesive rubber-plaster and left alone for several days.

Chrysarobin made into a solid stick of ointment, with beeswax, &c., as used by Brooke, affords a most efficacious method of applying the drug. The stick can be rubbed firmly into the patch, and the surface covered with plaster afterwards.

Pyrogallic acid is also a very efficient remedy, but considerable danger attends its use. The success of the treatment entirely depends upon the perseverance and thoroughness of the patient himself. Relapses are certainly very much less liable to recur if the tarry applications be continued long after the scales have disappeared. If the applications be promptly renewed as soon as the first signs of the characteristic papules or scales appear, the new attack can generally be rendered abortive.

Recently successes have been reported after exposure to the Röntgen rays. It must always be remembered that a small number of cases of general exfoliative dermatitis have been known to originate in a chronic psoriasis—a fact which should inculcate caution in the free use of stimulating applications in the treatment of psoriasis.

PURPURA

The term is applied to one sign or symptom which may be the result of several different pathological conditions. As ordinarily employed, the word 'Purpura' is used to designate the diseased states in which spontaneous or non-traumatic

extravasations of blood occur in the skin or the tissues beneath it, in the parenchyma of internal organs, or in the serous cavities, or where blood oozes from the mucous membranes of the body.

A classification of the different conditions in which hæmorrhages in any of the above-named sites constitute an obvious clinical feature is a matter of difficulty or of impossibility in the present imperfect state of our knowledge of the various diseased conditions of the blood. The difficulty may be partially met by a brief survey of the etiological factors usually regarded as causes of purpura, after which the clinical features of the most easily recognised varieties of the disease may be considered. These latter varieties are sometimes confusedly grouped together under the title of *idiopathic purpuras*, which include Purpura Simplex, Purpura Hæmorrhagica, Purpura Rheumatica, Henoch's Purpura, and Febrile Purpura.

Etiology.—The diseased or altered condition of the blood which occurs in the infective fevers leads in many instances to the production of hæmorrhages into the skin or other regions. Small-pox, typhus, measles, scarlatina, cerebro-spinal fever, plague, septicæmia and pyæmia, syphilis, malaria, and occasionally pneumonic fever, may so alter the composition of the circulating fluid that a purpuric condition results, which manifests its presence by hæmorrhage into the skin and in other regions. If the pathology of acute rheumatism and malignant or ulcerative endocarditis, which places these diseases in the same category as the infective fevers, be accepted, the rheumatic or arthritic form of purpura must be regarded as the result of a similarly acting etiological factor.

Poisons or toxins which are not the products of micro-organisms may induce purpura; the operation of this group of etiological factors is best seen in the hæmorrhages which complicate jaundice and snake-bites and in those which supervene after the administration of iodides, phosphorus, and other drugs.

Certain conditions, characterised by profound cachexia, as those seen in the primary anæmias, Hodgkin's disease, leukæmia, scurvy, tuberculosis, cancer, sarcoma, renal affections, and rickets very commonly lead to the production of purpura of various grades of severity.

Circulatory disturbances, as those resulting from venous stasis, thrombosis, valvular disease and the pressure of growths, and the temporary circulatory embarrassment occurring in epilepsy, pertussis, and angina may also cause extensive and numerous purpuric hæmorrhages.

A class of etiological agents, which for want of a better name is termed *neurotic*, has been long recognised; the best examples of this are seen in the purpuric extravasations which

occur in connection with the lightning-pains and joint-crises of ataxia, and the arthritic manifestations sometimes observed in various spinal diseases, hysteria, neuritis, and neuralgias.

If we exclude the rheumatic, all the above etiological factors—infectious, toxic, cachectic, mechanical, and nervous—are grouped together by Osler as causing various varieties of purpura, to which he gives the title 'Symptomatic Purpuræ.' There is, however, another well-marked condition in which the purpuric extravasations are secondary, and this is hæmophilia. In all the above diseased conditions the hæmorrhages may be looked upon as but a *symptom*, and though far from an ideal classification, with our present imperfect knowledge of purpura it is a clinical help to recognise the remaining forms of so-called spontaneous or idiopathic purpura, as separate *diseases* or as various phases of one disease.

Pathogeny.—In the symptomatic varieties evidently the primary change is one effected by an alteration of the blood by toxins produced within the body, or accidentally introduced from without. The altered blood, circulating in the small vessels, induces changes in their walls whereby, owing to diminished vitality, extravasations readily occur. The mechanical and nervous etiological factors probably act mainly upon the vascular walls through the vaso-motor nerve-supply. Some authorities, including Schwimmer, regard all purpuræ as of tropho-neuritic origin. The problem of the pathogeny of the idiopathic purpuræ is still awaiting solution. Probably further research will clear up the difficulty by the demonstration of specific micro-organisms. In the febrile form of the disease the etiological factor appears to be almost certainly a bacterium or coccus, though no specific microbe has been identified. It is a noteworthy fact that all these forms of purpura occur in the early years of life, the great majority of the cases being observed in children.

PURPURA SIMPLEX

Some authorities include this form under arthritic or rheumatic purpura, which tends to confuse and mislead. The disease occurs, as a rule, in children and young adults in whom no arthritic manifestations whatever are usually present. The etiology of the affection is unknown; often the child is in apparently perfect health when the eruption appears.

Symptoms.—In a small percentage of the cases there is some gastro-intestinal irritation manifesting itself by dyspeptic symptoms or by a smart diarrhœa of short duration, but, as a rule, the first indication of the disease is seen in the eruption.

This usually is discovered in the morning, and consists of true petechiæ or minute extravasations of a purple colour

beneath the epidermis. They vary from the size of a flea-bite to that of a sixpence, and their most important feature is that they do not disappear upon pressure ; moreover, they are never raised or elevated above the level of the normal cutaneous surface. They come out in successive crops, usually of symmetrical distribution, often appearing first on the legs ; they may ultimately cover the entire skin, and as they fade the purple colour gives way to the brown or yellowish-green coloration seen in the resolution of an ordinary bruise or ecchymosis. The spots are round, oval, irregular, or stellate in configuration, and do not increase in size unless by the coalescence or confluence with the petechiæ coming out in the next crop. There is no fever, itching, or sensory disturbance, and only occasionally are more or less anorexia, lassitude, and aching muscular pains complained of. If the mucous membrane of the buccal cavity be inspected, similar petechiæ may be often discovered. Vibices or small streaks of extravasation are often visible, and occasionally larger ecchymoses are observed in the subcutaneous tissue, probably the result of slight bruises ; but larger masses or hæmatomata are rare, as are also any hæmorrhages into the conjunctiva or from the mucous membranes, the eruption in simple purpura being in all typical cases confined to the skin. As already stated, joint-manifestations are entirely absent or only of the most trivial character. More or less anæmia may be present, but the red cells seldom fall below 4,000,000 in a c.mm. of blood. Complete recovery follows within a couple of weeks, as a rule, unless accidental bruises have caused larger extravasations : it is noteworthy that the condition is not liable to recurrences.

PURPURA HÆMORRHAGICA

This form is also known as Werlhof's Purpura, Land Scurvy, and Morbus Maculosus.

The disease is comparatively rare and, like simple purpura, is usually met with amongst young, otherwise healthy, subjects, though, more frequently than in the other varieties, a condition of previous debility has existed before the appearance of the skin-eruption. This latter may in no way differ from the petechiæ seen in the simple variety, though the spots are sometimes larger, but in a short time after the development of the eruption the characteristic feature of the disease shows itself in severe hæmorrhages from the mucous membranes. Epistaxis may be the first evidence of this tendency ; it may be severe and even fatal. The gums bleed profusely, hæmatemesis, melæna, hæmoptysis, hæmaturia, and metrorrhagia appear. Sometimes the hæmorrhages occur from the serous surfaces, causing death from pressure on the brain, or embarrassment of the heart and lungs when the thorax is

the site of the extravasations. Occasionally the bleeding may take place into the cerebral substance, into the retina, or into the parenchyma of the internal organs, and occasionally even into the substance of the tongue, causing dyspnoea and dysphagia.

The hæmorrhages may be frequent, and when coming from several surfaces at once a condition of anæmia and profound debility is rapidly produced. Hæmatomata, or large extravasations into the subcutaneous tissue or along the deep planes of the fasciæ or intermuscular septa, produce the same result. The patient, in apparently robust health upon the first appearance of the petechial eruption, may sink within a week from syncope after profound blanching caused by the loss of blood; but recovery may occur in the most hopeless cases, even when the number of red discs has fallen to almost one-fifth the normal.

In most cases the tendency to hæmorrhages exhausts itself, and within a fortnight recovery gradually commences. Werlhof regarded this as the rule in all cases, but the disease may be protracted over a much longer period. In a case recently observed by the writer the typical hæmorrhages continued, with but the briefest of intermissions, for more than nine months from nearly all the mucous surfaces in turn, death resulting from exhaustion long after the patient had rallied from the shock of what appeared to be almost certainly an extensive hæmorrhage into the peritoneal cavity.

In rare instances of this variety of the disease, occurring generally in young children, to which the title *purpura fulminans* is given, death has supervened within forty-eight hours, and even as early as the advent of the second day before mucous hæmorrhages had taken place. In addition to the above symptoms, fever is very generally present at some time or other during the attack, but it is seldom high, and as the anæmia increases a considerable degree of anasarca may be observed. This may even be extensive in some cases about the scrotum and penis, even before much blood is lost. A certain amount of anasarca on the dorsal aspects of the feet and hands is common.

PURPURA RHEUMATICA

Also known as Peliosis Rheumatica and as Schönlein's Disease, this is an acute disease characterised by arthritic pains and a purpuric eruption, which often exhibits multiform features.

The affection occurs at a later period of life than does simple purpura, the majority of the cases being met with about thirty; it is very rare before ten and after forty. The first symptoms usually observed are those of a mild attack of sub-acute rheumatism, in which there are more or less

severe pains in the wrists, ankles, knees, and elbows, and these joints may be tender and somewhat swollen; not uncommonly there is sore-throat and nearly always more or less fever. The eruption appears within a few days after the onset of the arthritic manifestations, commencing usually upon the lower extremities in the form of petechiæ; these sometimes appear in the immediate neighbourhood of the affected joints, and as a rule do not invade the trunk, though isolated petechiæ may be found anywhere. The joint-pains often diminish in intensity upon the development of the rash; but in some cases this appears first, the arthritic manifestations following. The writer has observed a typical severe acute rheumatism supervene in two cases after the complete disappearance of the eruption.

The eruption varies considerably. Petechiæ resembling those seen in simple purpura are always present and may constitute the entire skin-lesion, but nearly always raised papules or tuberculated patches of a bright-red colour, which may be as large as a shilling, develop; these soon become purple, and may closely simulate *erythema nodosum*, *E. papulatum*, or *E. tuberculatum*, but the elevations are, as a rule, uninfluenced by pressure. The petechial spots come out in successive crops, which may continue to appear during several weeks; they are symmetrical and pass through the usual changes of colour observed in the resolution of an ordinary ecchymosis. Relapses or recurrences are common. In some cases the above true hæmorrhagic skin-lesions are associated with distinct erythematous or urticarial rashes; these have been placed in separate groups under the titles *purpura urticans*, *erythematous purpura*, &c.

In the urticarial variety there may be large wheals associated with extensive œdema of the face, hands, and genitals. The erythematous form without wheals cannot be distinguished from *erythema exudativum multiforme* save by the prevailing true hæmorrhagic lesions always present. In rare cases the patches may become the seat of blebs—a condition to which the title *pemphigoid purpura* has been applied. This variety is only seen in its typical form in febrile purpura.

Pathology.—A close relationship to rheumatism is universally recognised owing to the presence of the arthritic manifestations and the occasional development of valvular lesions, chorea, pericarditis, and other complications of acute rheumatism. The closeness of this relationship is seen in those cases where attacks of peliosis rheumatica alternate with those of acute rheumatism. That there is also an affinity between the disease and *erythema exudativum multiforme* is highly probable, since joint-manifestations are almost constantly present in this latter affection, even though

there may be no true hæmorrhagic lesions, and occasionally a typical attack may be followed by acute rheumatism. The view that the skin-lesions in purpura rheumatica are the direct result of a poison circulating in the blood, and that this poison is a toxin produced by the rheumatic bacillus, is highly probable, though in many of the cases the fever may be trivial, and seldom is sweating present.

Diagnosis.—The occurrence of purpura (that is, a condition associated with a symmetrical skin-eruption of true petechial character) in a patient suffering at the same time from distinct pain, tenderness, and swelling in several joints leaves no doubt about the diagnosis. In those cases of *erythema multiforme* in which joint-pains are marked and the eruption is distinctly erythematous, the papules or tuberculated patches disappearing upon pressure, there need be little hesitation in excluding purpura. But if such cases be closely watched from day to day, often true petechiæ may be discovered; whether the presence of these be accidental or not, the diagnosis may be safely altered under such circumstances. It is of little importance to discuss whether such a case is to be considered as one of purpuric erythema or of true rheumatic purpura, the underlying condition being probably identical, though modified by some peculiarity of the patient.

Prognosis.—This is always good; rheumatic purpura tends almost invariably towards recovery, unless there be endo- or pericardial complications present. In rare cases, however, the purpura has been observed to develop into the hæmorrhagic variety of the disease, in which bleedings from the mucous surfaces may prove serious. In most cases relapses may be safely predicted sooner or later. These not infrequently occur if the patient begins to move about or expose himself to cold within a short period after the disappearance of the eruption.

HENOCH'S PURPURA

This variety of the disease usually attacks children, but, as in the last-described affection, adults also suffer, though much less frequently; thus it is very rare after thirty-five, and most of the cases occur between seven and fourteen. The most obvious clinical feature in Henoch's purpura, in addition to the symmetrical petechial eruption and joint-pains, is the presence of marked gastro-enteric symptoms. The illness begins like a typical rheumatic purpura in the great majority of cases, but after a few days intense abdominal pain of a colicky nature supervenes, associated generally with bloody vomiting and diarrhœa, in which much blood appears in the stools; these may consist entirely for a time of altered blood. These abdominal symptoms only very occasionally precede the joint-pains and eruption; they may all be present some-

times without the appearance of blood in either the vomit or motions. Of short duration, these attacks recur every four or five days, or at longer intervals of a week or more, for many months, the recurrent tendency constituting an important diagnostic feature in the disease. Epistaxis and hæmorrhage from the gums, and even hæmoptysis, may be observed. The urine is often bloody; there is more albumin present than is accounted for by the blood, but sometimes abundant albuminuria may be found without red cells being discovered on microscopical examination. The urinary symptoms and signs are thus indicative of a severe form of nephritis, which is liable to terminate in a fatal uræmia, though sometimes the renal affection resolves. Enlargement of the spleen has been detected during life and found after death, and necrotic foci have been observed in the congested gastric and intestinal mucosa. The petechiæ may be confined to the legs or to the legs and arms, but sometimes they involve the entire surface of the body, even including the face and visible mucous membranes. Often, as in severe peliosis rheumatica, the true petechial eruption is complicated with urticaria and large wheals, constituting the skin-condition recognised as purpura urticans. Sometimes the prevailing type of the eruption is erythematous, the exudative multiform character being well marked; but always in association with this erythema multiforme true petechiæ are present, and there is a marked tendency to local anasarca, constituting the condition recognised as *angio-neurotic œdema*. The joint-manifestations in many cases are but slightly marked, and sometimes throughout the attacks fever may be absent or trivial, and no sweating may be observed. Osler lays stress upon the varying characters of the eruption, which may differ greatly in the various attacks in the same patient from time to time.

Pathology.—The pathogeny of the affection is supposed to be allied to that of peliosis rheumatica; in a few cases the complications of acute rheumatism have been found present. Some authorities regard the symptoms as the result of vaso-motor disturbances, probably induced by the rheumatic or some unknown toxin.

Diagnosis.—The chief points have been already emphasised in the description; they may be summarised in the definition of a true purpura associated with arthritic manifestations, a varying superadded erythematous or urticarial form of eruption, associated always with recurring attacks of gastro-intestinal pain and hæmorrhages and frequently with serious nephritis. In one case recently under the writer's observation the clinical picture closely resembled that of *trichinosis*, the œdema about the eyelids and orbit being very well marked.

Prognosis.—The natural tendency of the disease is towards recovery, unless when severe renal changes occur, in which

case death from uræmia is not a rare event ; but prognosis is always most uncertain as regards the duration of the illness, which by frequent recurrences may extend over many months, or a few years in rare cases.

FEBRILE PURPURA

Under this title a fatal form of purpura is recognised which commonly affects young adults, the amount of pyrexia present being always much greater than that observed in the other varieties of the disease. The onset is often sudden and severe, though usually a history of a gradually increasing pallor may be obtained. The eruption consists of abundant crops of true petechiæ interspersed with somewhat raised purple patches, showing a tendency to the formation of pemphigoid bullæ, which upon bursting leave necrotic spreading ulcers. A similar necrosing or gangrenous action supervenes upon the petechial patches found in the buccal cavity, leading to the formation of extensive ulcers and sloughs upon the gums, inside of the cheeks, and tonsils, and even involving the laryngeal mucous membrane. The fetid breath and bleeding gums, with the rapidly increasing debility, profound anæmia, and tendency to heart-failure, may closely simulate the clinical picture of severe scurvy, but the gums are never swollen or spongy. The intensity of the anæmia, even when the only loss of blood is a trivial oozing from the gums, and the high degree of the pyrexia are in marked contrast with the condition seen in the other varieties of purpura. Joint-pains may be complained of, but these are usually entirely subjective, no swelling being visible. Though epistaxis is common and some bleeding from the pallid gums is almost invariably present, hæmatemesis, hæmaturia, and melæna are very seldom observed, and if met with are trivial in degree ; the spleen is usually enlarged, and retinal hæmorrhages are common.

Pathology.—The pathogeny of the affection is unknown, though a rapid toxæmia obviously seriously affecting the composition and constitution of the circulating fluid is probably the explanation of the serious anæmia, fever, and other signs of the disease. The toxin will very likely be proved to be of microbic production.

The prognosis is always grave ; Hawkins states that it is doubtful if recovery ever occurs. Death usually takes place before the termination of fourteen days, often within a week, the patient sinking from cardiac asthenia.

Treatment of Purpura.—In all the varieties, sympathetic or idiopathic, the patient should be compelled to rest in bed, unless in the mildest cases of the simple form. Posture has a marked effect upon all cases of mild simple purpura, the eruption sometimes entirely disappearing upon the patient

resting in bed for a few days, to return again after he commences to move about, especially if the petechiæ are confined to the legs. A generous diet of concentrated soup and milk foods is the best. The cause, when discoverable, should if possible be removed; but as this is seldom within the power of the physician, except in the symptomatic purpuras caused by mineral poisoning, he is compelled to treat symptoms as they arise upon recognised therapeutic principles or to resort empirically to the administration of drugs which experience has proved to be of any value. Of these so-called specifics turpentine, antipyrin, and nitrate of silver may be tried. The ordinary astringents, as tannin-containing drugs, ergot, lead, sulphuric acid, &c., are of no value and need not be relied upon to control bleeding. Chloride of calcium, so efficacious in the treatment of other hæmorrhagic conditions, is usually disappointing, but it should always be administered.

In the hæmorrhagic form Fenwick and Parkinson have recorded benefit from rectal injections of anti-streptococcic serum. In the rheumatic variety salicylates, upon theoretical considerations, are clearly indicated; but most observers agree that they have no influence upon the blood-condition. Purpura simplex will resolve usually without any drug-treatment, but a good routine will be to administer a mixture containing iron or arsenic and chlorate of potassium. Iron and arsenic are always indicated in the treatment of the resulting anæmia in all hæmorrhagic cases. Strychnine only can be relied upon as a temporary measure in combating cardiac failure.

PYÆMIA

This condition, mainly of surgical interest, requires but a brief notice, especially as its local manifestations have been already dealt with in the description of such affections as abscesses of the liver and brain, ulcerative endocarditis, erysipelas, empyema, &c. The affection is best considered in conjunction with Septicæmia, with which it is so closely allied, but from which clinically it is sharply defined; also its relationship to ordinary septic infection or Sapræmia may be referred to.

SAPRÆMIA

In *sapræmia* a soluble chemical poison or toxin, the product of pathogenic microbes, is absorbed, and, finding its way into the blood, produces the characteristic symptoms of a general septic intoxication. The condition is seen in its most typical form in diphtheria, in which the bacilli, multiplying in the false membrane upon a mucous surface, manufacture a toxin, which, upon being carried by the circulating fluid to the

different tissues, selects those which in some way are most susceptible to its subtle action, by means of which certain specific effects are produced upon the nerves and cardiac muscular fibres. The selective action is in all probability determined by a chemical union between the toxin and the protoplasm of the tissue affected. The progress of pharmacological research, which has demonstrated the close relationship between the chemical *constitution* of a body and its physiological action, is a valuable assistance to the interpretation of the phenomena witnessed in many diseased processes. The epoch-marking researches of Crum Brown and Fraser upon the action of strychnine on the constituents of the spinal cord proved that on alteration of the constitution of the strychnine molecule by the introduction of methyl a paralysing instead of a convulsive action could be produced at will. If this law of chemical constitution being the chief factor in physiological action be accepted, it follows that the symptoms and pathological changes observed in the majority of diseases may, without unduly straining the application, be regarded in the light of a chemical result. Such a conception will widen the horizon very considerably and assist the physician in interpreting the manifold phenomena of symptomatology in such septic intoxications as diphtheria, tetanus, pneumonia, &c., and all the bacterial diseases causing fever, &c. The study of the symptoms of such affections may, then, be regarded from the standpoint of an ordinary pharmacological problem, in which, for example, the selective action of the tetanus-toxin upon the spinal neurons may be contrasted with the actions of strychnine, alcohol, chloral, or other organic toxic substances. The recognition of the probability of these phenomena being the result of a chemical process, though not necessarily capable of demonstration, helps the elucidation of the problem considerably. Moreover, it assists in the better understanding of the means used in the antagonising of the symptoms by antidotal agents. The theory of the presence of side-chains, introduced by Ehrlich in his attempt to explain the action of the antitoxin of tetanus, exhibits a tendency towards the same direction of thought.

Sapremia, typified in diphtheria as being the result of the action of a chemical poison absorbed from the focus of disease upon the surface of the mucous membrane, is not to be confused with the more complex condition to be next referred to, in which the bacteria themselves are admitted to the blood. Though in diphtheria sometimes the microbes do gain admission to the blood, their entrance is more or less accidental, and their presence in the circulating fluid plays no part in the phenomena of the septic intoxication. The symptoms can all be exactly reproduced by the injection of the sterile diphtheritic toxin in animals, especially when this is

manufactured by cultivation of the specific bacillus in certain alkali albumin media, as effected by the method adopted by Sydney Martin. The same remarks apply to tetanus-intoxication, erysipelas, and pneumonia, in which the micro-organisms usually play no part except in the manufacture of their toxins at the sites of the local infection.

SEPTICÆMIA

This is the condition in which not only the specific chemical poison or toxin is circulating in the blood, but the bacteria or other microbes are themselves present and capable of multiplication in the circulating fluid or tissues. The main difference between the two conditions obviously lies in the capability of reproduction and the power of the multiplying organisms to produce continuously increasing amounts of the intoxicant. Septicæmia may exist without a local site or focus of infection, but as a rule such is present, and the first stage of the illness may commence in a typical sapræmia as above defined. After a varying time, in which the soluble toxins are alone absorbed, the bacilli or cocci find entrance to the blood and tissues, in which they increase and multiply, and true septicæmia results. This is often seen in the septic disease—erysipelas—after the introduction to the blood of the streptococci which cause the skin-lesion. It is also commonly seen in diphtheria, where the true toxin of the specific bacillus is reinforced by an invasion of streptococcic and staphylococcic germs in the circulating fluid. These latter lift the multiple infection out of the category of a sapræmia into that of a true septicæmia. The most typical instance of a simple septicæmia, however, is seen in the inoculation of wounds, either accidental or surgical, by pathogenic organisms, or in the inoculation during labour of the microbes which produce puerperal fever. The gonococcus, typhoid bacillus, the pneumococcus, and the anthrax infection, at first local, may also eventuate in a true general invasion. Septicæmia may result from the introduction into the blood and tissues of saprophytes—viz. organisms which in the normal healthy condition exist in the body, but become under certain conditions pathogenic. This is seen in the septicæmic invasions arising from the bacillus coli communis, staphylococcus pyogenes aureus, and streptococcus pyogenes. The conditions permitting of such invasions are probably due to the suspension of the activity of the normal phagocytic mechanism. Some of these examples constitute the so-called *cryptogenetic septicæmia*, in which no local foci of infection can be determined during life or after death. They are usually produced by streptococcus pyogenes, and often occur as terminal infections in patients whose natural resistance is diminished by the presence of some debilitating disease.

PYÆMIA

This is the still graver condition in which the infecting microbes pass out from the blood into the tissues and form multiple abscesses—the so-called metastatic or embolic abscesses. The suppuration is usually determined by the detachment of portions of infected material from the original thrombotic focus in a vein near the seat of injury, which, being carried to a distance, act as emboli blocking the internal veins. The micro-organisms establish by their development a thrombotic process—a new purulent phlebitis—which eventuates in an abscess through the breaking down of the tissue in the immediate neighbourhood of the obstructed vein. In the metastatic abscesses which occur in ulcerative endocarditis the arteries receive the infected emboli from the vegetations on the cardiac valves, so that the term '*arterial pyæmia*' is applied to the condition.

In pyæmia the organisms are various; most frequently they are found to be streptococci and staphylococci, but the pneumococcus, the gonococcus, the bacillus of influenza, ulcerative endocarditis, typhoid fever, *B. coli*, *B. proteus*, and *B. pyocyaneus* are all capable of producing infective thrombi which induce a purulent phlebitis at distant parts.

In most cases of pyæmia the infective focus can be recognised: frequently it is an open wound, sometimes an abscess in the middle ear, in the appendix region, in a lymphatic gland, or in the prostate, or an acute necrosing process in bone; sometimes a gonorrhœa or an old empyema may be the origin. In those cases where no obvious focus may be visible or palpable, careful investigation may reveal symptoms of an ulceration in the urinary or gastro-intestinal tract or in the endocardium or in the biliary passages in connection with gall-stones. Where no focus is discoverable the term *idopathic pyæmia* is applied; such cases may possibly be examples arising out of the cryptogenetic septicæmia already mentioned. Thus, whilst a simple sapræmia may pass into a true septicæmia, the latter may eventuate in pyæmia, though the majority of cases of pyæmia exhibit early tendency to the formation of metastatic abscesses, as seen by the succession of severe rigors, high fever, and profuse sweating from the beginning.

The three conditions—sapræmia, septicæmia, and pyæmia—are too frequently regarded as phases of so-called 'blood-poisoning,' but the conditions should, when possible, which is generally the case, be clearly recognised as essentially different from both the pathological and clinical standpoint. A sapræmia, for example, is usually speedily brought to an end by the removal of the external focus containing the infective organisms; in septicæmia, the microbes having

already gained an entrance into the circulating fluid, slight improvement only may be expected to follow local treatment, and in pyæmia the only hope usually lies in preventive treatment.

The multiple abscesses of pyæmia may occur in any region of the body; in many instances their distribution may be anticipated or accounted for by a knowledge of the relationships of the primary focus with the circulation. Thus, whilst in most cases the lungs are involved, in those instances of infection arising in the gastro-intestinal tract the liver is the chief seat, whether a suppurative pylephlebitis is present or not; such cases are frequently referred to as *portal pyæmia* (see under 'Thrombosis, p. 1695'). In the ordinary occurrence of pyæmia, arising from skin-wounds, the infected emboli detached from the inflamed veins at the injured area are brought to the lungs, where wedge-shaped infarcts form, which induce a rapid pneumonic process ending in multiple abscess, often associated with pleurisy and pericarditis. Though even the minutest emboli, which consist of blood-clot in which the micro-organisms are entangled, are not capable of passing through the pulmonary capillary system, there is no difficulty in realising how the microbes themselves may pass through the lungs and form foci of suppuration in the spleen, kidney, brain, joints, or other portions of the body.

In the pyæmia resulting from acute osteomyelitis, though the lungs are usually invaded, the kidneys appear to suffer most frequently from the metastatic abscesses. The heart-muscle not unfrequently is the site of the suppurative process, and endocardial vegetations are often formed which still further spread the mischief throughout the body. In pyæmia which runs a slow course the multiple abscesses are often confined to the joints or planes of areolar tissue. In the pyæmia resulting from ear-disease secondary thrombosis of the cerebral sinuses and veins arises from extension, and metastatic abscesses in the lung nearly always occur from the detachment of the infected thrombi, and empyema is common.

Symptoms.—The first evidence of the supervention of pyæmia in a case of external wound often follows the milder symptoms of septicæmia, but the onset may be abrupt and sudden, being ushered in by a severe prolonged rigor in which the temperature may register 105° or more; the shiver is followed by copious and often drenching perspirations, like those witnessed in malaria. These chills are liable to occur daily, or several times a day in severe cases, but they exhibit no rhythm or regularity, and the temperature falls often below the normal between the rigors.

The usual concomitants of severe pyrexia are present—viz. thirst, dryness of the tongue, anorexia, rapid breathing, &c.;

often, vomiting and sometimes, diarrhœa are severe ; marked emaciation and profound prostration rapidly supervene. The heart shows weakness at an early stage, the pulse becoming quick, small, and thready. An important diagnostic sign sometimes present is the appearance of fugitive patches of erythema in different regions of the skin. The physiognomy is often highly characteristic, even before local symptoms point to the site of the metastatic abscesses. The countenance early becomes pallid, but soon afterwards sallowness or a mild degree of jaundice supervenes upon the underlying anæmia, and the breath sometimes exhales a heavy sweet odour, somewhat suggestive of diabetes.

The clinical picture varies with the seat of the multiple abscesses as these begin to produce local symptoms, and the entire symptomatology of the affection may be marked by the disturbances caused by the primary affection—as, for example, when this is an otitis associated with meningitis, or an ulcerative endocarditis, pyelitis, or intestinal ulceration. The pulmonary symptoms and signs are those most commonly present, but they are often vaguely marked ; there are dyspnœa, cough, rusty or purulent sputum, more or less dulness, bronchial breathing, and râles of all kinds. Sometimes, however, the signs of a profuse purulent effusion may suddenly appear with typical distinctness. When the liver becomes riddled with multiple suppurating areas, more or less tenderness and enlargement may be obvious, but severe jaundice is seldom met with. Either by the coalescence of several small hepatic abscesses, or by the production of one larger than the others and situated near the hepatic margin, fluctuation may be present, but this is not by any means a common phenomenon.

The spleen may often be found enlarged, tender, and soft, and sometimes it becomes the seat of severe pain, and, as also in the case of the liver, friction may be felt or heard over its site. The kidney-signs are usually vaguely marked, but pus, blood, and the micro-organisms of the disease may sometimes be detected in the urine.

The joints and cardiac area should be carefully explored from day to day. It is often astonishing to find a large joint, like the knee, changed into a bag of pus within a single night after a severe rigor, and it is not uncommon to witness extensive pericardial and endocardial mischief develop within a few hours. Leucocytosis is always present in pyæmia, and occasionally the specific organisms have been detected in the blood. The duration of the disease is often short, death occurring from coma supervening rapidly upon a typhoid state within a week or less of the initial rigor. In the rarer chronic cases, where the multiple abscesses usually show a tendency to avoid the visceral organs, the disease may

last for many months, as one joint after another becomes the seat of the suppurative process. Often this arthritic suppuration is unassociated with local pyrexia, so that the 'cold' type of abscess is said to exist, and this is especially true of the suppurations which occur in the areolar tissue.

Diagnosis.—The presence of a succession of rigors, with sweating, and a markedly high but very irregular temperature, occurring in a patient suffering from an open wound or recovering from parturition or abortion, usually leaves no doubt about the diagnosis. The interpretation of the *initial* rigor in the case of closed wounds is an important matter. In pyæmia the superficial wound, at the time of the first onset, may be found to have changed its healthy appearance, the pus ceasing to be 'laudable,' a thin ichor, sometimes with an offensive odour, replacing the abundant healthy secretion. If, in a closed wound, upon inspection a high tension, caused by a pent-up condition of the pus, be detected, even a severe and long-continued rigor should not be accepted as evidence of the onset of pyæmia. The rigor in this case will probably be due to the temporary retention of the secretion and the absorption of toxins—*i.e.* the case is one of *sapremia*, or possibly of *septicæmia*, and the free evacuation of the discharge may be followed by rapid disappearance of all fever, &c., but the recurrence of the rigors may safely be accepted as evidence that metastatic deposits are being laid down in distant organs, demonstrating the true pyæmic nature of the affection.

In severe *septicæmia*, the fever may remain continuously high; delirium, when present, occurs early, and usually there are no rigors after the initial chill, contrasting markedly with the highly remittent, intermittent, or irregular type of fever, frequent rigors, profuse sweatings, and remarkable emaciation and prostration of true pyæmia. These essential differences in the two conditions must always be borne in mind in the diagnosis of all doubtful cases, and evidence of metastatic deposits carefully sought for.

The only practical difficulty in the recognition of the disease is to be expected in the cryptogenetic cases where no obvious primary focus can be detected, but these are exceedingly rare. An otitis, gonorrhœa, septic cystitis, pyelitis, gastric or intestinal ulcer or abscess in any region of the body must not be overlooked. Occasionally a deep suppurating focus in bone may be discovered by its deep tenderness, where no skin-implication has occurred to excite suspicion. The difficulty in recognising some forms of *ulcerative endocarditis* has been already dwelt upon in the description of that disease. The causal micro-organisms of pyæmia and of *septicæmia* are now easily demonstrated by the new method of blood-examination to be mentioned under the diagnosis of typhoid fever.

Malaria is only likely to be thought of in malarious districts, where its prevalence is recognised; the irregularity of the fever and rigors when the temperature-chart extends over a few days is obvious, and when a doubt remains the worthlessness of quinine demonstrates the pyæmic nature of the case. The form of so-called malarial ulcerative endocarditis may exactly simulate for a time a true quotidian (double tertian) malaria. The arterial plugging in such cases is pathognomonic.

Typhoid fever is sometimes differentiated with difficulty in the chronic variety of pyæmia where no metastatic deposits can be located. Attention to the following points will prevent mistakes. In typhoid, leucocytosis is not marked and a positive Widal reaction is present, though the absence of the characteristic eruption and the enlargement of the spleen may be suggestive of pyæmia; moreover, the continuous type of the temperature is usually characteristic.

Acute miliary tuberculosis may for a time closely simulate pyæmia when it occurs in the very rare form in which numerous chills follow each other several times in a single day. The cyanosis and dyspnoea are usually well-marked. Such cases rapidly pass into one or other of the three types of tubercular fever—viz. the pulmonary, typhoid, or meningeal.

Obstructive jaundice, accompanied by septic cholangitis, is a condition closely allied to and sometimes passing into pyæmia; the differential diagnosis can only be made after a careful survey of the history and close observation of the progress of the disease. The deep jaundice itself cannot be mistaken for the icteroid tinge of pyæmia, but a true pyæmia may supervene after ulceration of the gall-bladder or of the walls of the bile-ducts, in a patient who has been already severely jaundiced.

Acute rheumatism is not likely to be mistaken for pyæmia unless when the latter occurs in connection with recent parturition, or with acute necrosis in the neighbourhood of joints, or with a general infection from the gonococcus. The history in each case will determine the diagnosis when taken along with the characteristic distribution of the joint-affection in true rheumatism, which is very different from the apparently haphazard implication of the joints in all forms of pyæmia.

Prognosis.—In pyæmia this is grave and invariably more serious than in septicæmia. A fatal result is to be confidently expected in all acute cases and where the metastatic abscesses have invaded the viscera. The chronic form occasionally ends in recovery, even after several joints have been destroyed.

Treatment.—In sapræmia and septicæmia surgical measures are urgently indicated; and though in the latter condition the micro-organisms have already been admitted to the circulation, some benefit may often be obtained by the free

evacuation of all pus, the establishment of efficient drainage, and the liberal and judicious employment of antiseptics. These measures may also be regarded as constituting the preventive or prophylactic treatment of pyæmia. The saturation of the patient's system with large doses of iron alone or in combination with quinine has also a valuable prophylactic action in all septic states.

In chronic pyæmia the free incision of all affected joints, of the pleural, and even of the pericardial sac should be resorted to, and occasionally success follows; but in the acute form surgical interference is as a rule worse than useless. The fever, sweating, and rigors are best treated symptomatically by large doses of quinine.

Antistreptococcic sera have as a rule proved valueless.

PYELITIS

As its name implies, this condition is the result of inflammation attacking the pelvis of the kidney or the calyces. Other diseased states of the kidney are so frequently found associated with pyelitis, either in the relation of cause or effect, that it is hardly possible to describe this condition without frequent reference to them. The present article will therefore also include the consideration of such affections as:

(1) Pyelonephritis—a condition in which pyelitis is associated with inflammation of the renal substance.

(2) Ascending Nephritis or Acute Suppurative Nephritis or Consecutive Nephritis or Surgical Kidney, a condition in which suppurative inflammation of the pelvis of the kidney and of the true renal substance is an extension upwards of an inflammation of the bladder, prostate, urethra, or ureter.

(3) So-called Strumous Pyelitis—a condition due to the inflammatory action produced by the tubercle bacillus.

(4) Pyonephrosis—a condition which may be the direct result of any of the before-mentioned affections when, owing to blocking of the ureter, pus accumulates in the renal pelvis to the extent of forming a palpable tumour.

Etiology.—The causation of pyelitis is very varied, the most obvious factors being: (1) Direct irritants, as calculi, uric acid, gravel, or parasites and their ova, which by their presence produce inflammation of the pelvic membrane.

(2) Irritants excreted from the blood after their absorption from the stomach, as cantharides, copaiba, cubebs, and turpentine, may produce severe catarrhal pyelitis on their elimination by the renal tract, in which case renal congestion and strangury will also result.

(3) The poisons which produce small-pox, typhus and typhoid fevers, dysentery, cholera, and other microbic diseases,

(4) Trauma and exposure to cold in some cases may be sufficient to excite a transient pyelitis.

(5) The mechanical obstruction produced by the displacement of a freely movable or floating kidney, when the ureter becomes twisted or acutely bent, though it usually causes hydronephrosis, which may become purulent, sometimes excites symptoms of smart pyelitis.

(6) The most frequent cause of pyelitis is an extension upwards of the inflammation of the bladder or lower urinary tract, which is the result of stone in the bladder, enlarged prostate, stricture, paraplegia, or other cause of obstructed flow. The inflammation extends from bladder to renal pelvis and calyces by the ureter, and it may remain a pyelitis for an indefinite time, or it may lead to suppuration of the renal tissue, when the case will be better styled one of Consecutive or Ascending Nephritis.

(7) The degree of pyelitis which is secondary to some forms of Bright's disease and diabetes is usually so indefinitely marked by symptoms that it is but of pathological significance.

(8) Micro-organisms of various kinds play an important rôle in the production of pyelitis. Once admitted to the bladder, the pyogenic bacteria increase and multiply, and readily find their way along the ureters, especially when there is obstructed flow or a large quantity of residual urine constantly in the bladder. By some authorities all forms of pyelitis are considered to be of bacterial origin, and the previously mentioned causes are merely regarded as but the etiological factors which weaken the local resistance and prepare a way for the microbe. The truth of this view is obvious in those cases where severe pyelitis rapidly follows the use of non-sterile catheters or bladder-sounds.

The bacillus coli in intestinal obstruction soon assumes virulent forms and, entering the blood, ultimately may excite pyelitis; and this also may be the explanation of the pyelitis met with in the exanthemata, though usually it is the toxins which are credited with the irritating properties. It is well to remember in this connection that the bacilli of typhoid fever may be found in the urine many months after convalescence. The pyelitis resulting from the tubercle bacillus will be referred to later on.

The conditions which produce passive congestion of the kidney, as the pressure of ovarian and other tumours, cancer, valvular disease, pregnancy, &c., are by some regarded as productive of pyelitis by preparing a nidus for micro-organisms which, reaching the renal pelvis from the bloodstream, are in the normal condition swept out of the body in the urine.

Pathological Anatomy.—This will vary with the cause. In acute cases of simple pyelitis the ordinary appearance of

a mild catarrhal inflammation of a mucous membrane is all that is present. The lining membrane of the renal pelvis is red and swollen and numerous ecchymoses may usually be seen in its substance, whilst the surface is covered over with a layer of mucus containing the characteristic imbricated epithelial cells and some blood-discs. Where the inflammation is the result of the administration or absorption of drugs like cantharides, a thin fibrinous or croupous membrane has been found on the inflamed surface. In those cases occurring during the exanthemata the membrane has been described as diphtheritic in character, and a similar appearance is constantly found when the urine in the pelvis and bladder has been ammoniacal.

In the chronic cases the colour of the mucous surface is paler, and there is much thickening of the membrane extending to the deeper layers of the submucous tissue; the surface is rough, and large veins are visible upon it. Ulceration is not uncommon in calculous pyelitis, and pus is always present in the chronic forms.

With the above-mentioned changes will be found those characteristic of pyonephrosis when the ureter has been blocked, and of suppuration of the renal substance when pyelonephritis is present. Evidence of interstitial nephritis without any suppurative foci is often obvious in some chronic cases.

All the above changes may be present in the pyelitis which arises from obstruction in the lower urinary tract, and it is an important fact that this form of ascending nephritis is often *unilateral*, or much more advanced on the one side than on the other. The healthy kidney not infrequently becomes the seat of hypertrophic changes. The ureter on the affected side commonly presents evidence of chronic inflammatory changes.

Symptoms.—These depend largely upon the cause of the pyelitis; in the calculous form severe lumbar pain and tenderness over the kidney are more or less constantly present, at times presenting all the characteristics of renal colic already described. Smart fever, rigors, vomiting, and sweating supervene when the ureter becomes blocked, which pass away entirely should the concretion find its way into the bladder. Pyonephrosis, with the formation of a renal tumour, develops and becomes permanent when the ureter is impervious; more frequently the condition produces intermittent symptoms as the purulent accumulation is permitted to pass by the incomplete obstruction.

In the pyelitis produced by irritating drugs of the cantharides class the symptoms will be mainly those of the irritation caused by the associated bladder-symptoms, which may entirely mask the signs and symptoms of the renal

pelvic trouble; and the difficulty in recognising these is increased still further by the acute glomerulitis, usually present, which may cause intense hæmaturia.

The pyelitis, secondary to the exanthemata, rarely produces marked symptoms, the only evidence of its existence being found in the urine, which, in addition to pus, mucus, and epithelium, may contain shreds of the fibrinous membrane.

The *urine* in all cases of pyelitis presents more or less definite changes, and the diagnosis mainly depends upon the result of its microscopic and chemical examination. Pyuria is a constant sign in all cases, save in some instances of the acute variety, caused by irritant drugs. The pus varies greatly in amount; in the acute form it is usually scanty and always mixed with mucus, many blood-cells, and renal epithelium from the pelvic surface; the urine is generally acid. In chronic cases the quantity of pus may be very great, and the writer has seen copious purulent discharge remaining constant for very long periods without any other symptom or sign of the disease.

In unilateral pyelitis of long duration the pus may suddenly disappear from the urine, which then presents the typically normal characters. This is caused by blocking of the ureter on the affected side, the pyuria as suddenly reappearing on the obstructing clot or concretion being swept downwards. This intermittent pyuria is very characteristic, and may be a marked feature in the case even when no signs of pyonephrosis are present; it never occurs in chronic bilateral pyelitis.

The epithelium found on microscopic examination, though strongly suggestive of pyelitis, is not pathognomonic; the imbricated club-shaped or caudate cells from the renal pelvis cannot be differentiated from the similar cells derived from the bladder and ureter.

Crystals of uric acid or oxalate of lime may give indications of the nature of the irritant causing the pyelitis, and occasionally the identification of hydatid hooklets or cysts, or of the ova of the Bilharzia or other parasite, will demonstrate the nature of the case. As already stated, the discovery of croupous or fibrinous membrane in the urine may be expected in the pyelitis of typhoid and other fevers; diphtheritic membranous shreds are usually found when the urine in the pelvis of the kidney has undergone alkaline or ammoniacal change. Even in long-standing cases the reaction of the urine may remain slightly acid, a point of considerable value in the elimination of bladder-disease, in which the urine will generally be found to be ammoniacal; but decomposing urine in the renal pelvis will also exhibit alkaline reaction, and this decomposition will depend upon the nature of the microbe, which is often staphylococcus pyogenes.

Albuminuria is always present, but in pyelitis, where the renal substance is not involved, it is invariably slight, being strictly in proportion to the amount of pus-cells present. When there is a question of the identity of the cells being those of pus, a filtered specimen of the urine will settle the diagnosis; if no trace of albumin is present the cells may be pronounced not to be pus-cells, but normal leucocytes or mucus-corpuseles. The purulent deposit becomes tenacious and stringy on the addition of liquor potassæ, and this is the condition of the deposit in all cases where the purulent urine is passed in an ammoniacal state. Albumin becomes copious in proportion to the amount of nephritis or of injury to the renal parenchyma.

The amount of urine is usually increased in chronic forms of pyelitis, and its s.g. proportionally diminished. Where the renal substance is involved there may be great increase in the amount of water, since this is not absorbed during its sojourn in the tubules of the medullary portion of the kidney. Anuria is liable to occur in the acute cases where both kidneys are the seat of pyelitis, and it occasionally supervenes without obvious cause in unilateral cases where the irritation has become acute from some accidental change.

Where the pyelitis is of the ascending type, arising in the extension of the bladder-inflammation caused by obstructed flow, the symptoms and signs are masked by those of the cystitis; but it is important to remember that a pyelitis of renal origin, when acute, may first manifest its presence by frequency in micturition, which is probably a reflex phenomenon. Moreover, the reaction of the urine in bladder-cases is almost always alkaline, often ammoniacal, and fetid.

PYELONEPHRITIS

This may be regarded as present when, in addition to the pyuria and other symptoms, there are added those of suppuration in the kidney. Fever of the intermittent type usually sets in with rigors and sweating, indicative of multiple abscesses in the diseased organ, and the case soon assumes a more serious aspect, pyæmic deposits sometimes occurring in other organs, or a typhoid condition supervening, owing to the absorption of decomposed products, to which latter state the term 'ammonæmia' has been applied. Occasionally the abscess bursts into the renal pelvis, with temporary relief, or it may discharge outside the kidney, and the course may be then indefinitely prolonged, the sound organ becoming the seat of amyloid degeneration or chronic interstitial nephritis. Lumbar pain, renal tenderness and swelling, with increase of temperature, supervening upon an old pyelitis in which the ureter is not blocked, should lead to suspicion of the advent of pyelonephritis.

ASCENDING or SUPPURATIVE NEPHRITIS

This is included in the previous description. It is the term employed when the surgeon wishes to designate a suppurative condition of the kidney—viz. a pyelonephritis which has originated in inflammation of the lower urinary tract. The usual course is for the pyogenic bacteria to extend from the septic bladder along the ureter. After establishing a pyelitis they enter the collecting tubes and find their way along the finer tubules; by the lymph-channels they also reach the interstitial tissue, causing numerous circumscribed abscesses throughout the cortical and medullary substance. Sometimes the extension of the bladder-inflammation leads to a chronic pyelitis, which may eventuate in a *chronic interstitial nephritis* without pus-formation. The final change being that of contraction of the kidney caused by the shrinking of the new connective-tissue formation, the condition contrasts markedly with the dilated pelvis and calyces and with the often very widely distended ureters which are obvious features in the obstructed cases so often associated with pus. It must always be remembered that the suppurative nephritis, though originating in bladder-mischief, may be confined to one kidney.

STRUMOUS PYELITIS

This is the old name given to *chronic* or *caseous tuberculosis* of the kidney. The affection may begin in a kidney which has received its infection through the blood from some other organ or tissue, as from an old infected lymphatic gland, or it may be an extension upwards of the tubercular process from the testicle, epididymis, seminal vesicles, prostate, or bladder. The most frequent form is believed to be the so-called primary renal tuberculosis, which begins in the papillæ and extends downwards, involving the calyces, renal pelvis, ureter, and bladder. The disease is most common in males and in middle-life and is often unilateral, contrasting with acute miliary tuberculosis of the kidney, which is part of a general dissemination, occurs at an early age, is little influenced by sex, and is always bilateral.

Pathological Anatomy.—From the papillæ the tubercular process invades the pyramids and pelvis, and steadily extends through the renal substance outwards till the capsule is reached and the entire organ is destroyed. The tubercles coalesce, necrosis and caseation occur, leading to the formation of cysts or cavities filled by cheesy masses, which still further break down till they become of almost fluid consistence; or, their liquid parts being absorbed, the cavity becomes filled with firm putty-like material in which lime-salts are deposited, the whole forming a mass like mortar,

surrounded by the indurated and invaded fibrous capsule of the kidney.

During the process detritus from the parenchyma or from the pelvis of the kidney finds its way into the ureter, which may become blocked, leading to hydro- or, more frequently, pyonephrosis, with the formation of a large tumour. The ureter itself suffers in the tubercular invasion; it becomes thickened and its mucous membrane, like that of the renal pelvis, becomes the seat of ulceration. Gradually the bacilli invade the bladder, prostate, epididymis, or seminal vesicles. This invasion may occur through the stream of urine, but when the bladder is involved the tubercles may sometimes be observed to be deposited beneath an unaffected mucous membrane, suggesting an extension by means of the blood-stream. Ultimately the abdominal glands, peritoneum, and other organs become affected by metastasis. In the ascending form of the disease, where the kidney and pelvis become affected secondarily from the bladder or testicle, the bacilli probably find their way upwards by means of the blood or lymphatic stream, and not along the ureter against the descending urinary current. The opposite kidney is usually enlarged; it may be the seat of compensatory hypertrophy, but more frequently there is evidence of some tubercular pyelitis in it also.

Symptoms.—Occasionally these may be absent till the presence of a pyonephrosis leads to the discovery of the unsuspected tubercular affection. Sometimes an autopsy reveals an old obliterated organ whose place is occupied by a mortar-like mass of cheesy matter impregnated with calcareous material in a subject where no urinary symptoms had been complained of during life.

The first symptom to make its appearance may be frequency in micturition, soon followed by pus in the urine; at this stage the affection is liable to be mistaken for bladder-trouble, long before any secondary vesical invasion has occurred, the pollakuria being the result of reflex action originating in the renal pelvis. More frequently the prominent and persistent sign of the disease is pyuria, the pus being commonly accompanied by the presence of small quantities of blood and small masses of detritus or shreds of renal or mucous tissue, which under the microscope will reveal, after suitable staining, numerous tubercle bacilli. Casts are not usually present, but in one case under the writer's care, where the renal affection followed upon a tubercular orchitis, in a stained slide a well-formed cast of a uriniferous tube was discovered by Lorrain Smith, containing in its interior several tubercle bacilli.

Albuminuria is constantly present, unless in the rare cases where the ureter is early obliterated or blocked. Usually the amount of albumin is slight and is derived mainly from the

pus or blood, but repeated examinations will discover that there is more albumin than these will account for; and in some specimens of the urine it may be found in fair amount during a transient absence of the pyuria and hæmaturia. A free albuminuria may be the first evidence of lardaceous change in the opposite kidney.

Hæmaturia is rarely copious and prominent; it is more common in the early stages, and is not infrequently entirely absent throughout. Pain in the lumbar region is the rule, and tenderness is not often absent; sometimes renal colic precedes the discharge into the bladder of detritus, cheesy masses, or calcareous material. A tumour in the renal region may be found in most cases towards the end of their course, and, like the hydro- or pyonephrosis of calculous pyelitis, it may periodically discharge its contents into the bladder and disappear for a time.

Fever of an irregular or intermittent type is often present, and sweatings following smart rigors are common when the ureter becomes blocked. The advent of pyelonephritis at an early stage is marked by the fever of suppuration and the deepening of the constitutional weakness and emaciation.

The signs and symptoms of tuberculosis of other organs are rarely wanting in the late stages, and death may occur from pulmonary phthisis or tuberculosis of the intestinal tract or from uræmia.

The results of cystoscopic examination will be discussed in the section dealing with Diagnosis.

PYONEPHROSIS

This is the condition in which there is dilatation of the renal pelvis and calyces, with flattening of the papillæ, wasting, and final disappearance of the medullary and cortical parts of the kidney, the place of the organ being filled by a large sac or cyst, with thick and irregularly bossed walls, corresponding to the renal capsule and filled with pus. The cyst is often divided by fibrous bands or trabeculæ into numerous chambered cavities, which usually communicate with each other. Where the pus-formation has supervened upon a long-standing hydronephrosis, the cyst or sac may represent a thin and smooth-walled bag of very large dimensions, as described under 'Hydronephrosis,' p. 664. The contents may consist of caseous material, with calcareous deposits or putty or mortar-like masses, with variable proportions of pus and urinary liquid. Not infrequently the contents of the sac may be found in a state of putrefaction, especially when the bladder has been the seat of dilatation from obstructed flow with putrid residual urine.

When the condition supervenes upon a hydronephrosis which has been caused by renal calculi, these may be found

in numbers in the sac, and one of the stones may be found implanted in the ureter.

The causes which produce hydronephrosis have been already stated; all of these may end in the production of pus, when the case will then pass into one of pyonephrosis. Thus calculi, tumours in the pelvis or abdomen, pregnancy, floating kidney, the blocking of the ureters by bladder-disease, parasites, &c., may cause distension of the renal pelvis and absorption of the secreting structure by the pressure of the watery fluid, which latter may become purulent on the super-vention of inflammatory action and the introduction of pyogenic organisms. All the causes which produce pyelitis are also liable to eventuate in a pyonephrosis without the intervention of a hydronephrosis, since in most cases the process is purulent from the beginning; and this is especially true of the pyelitis caused by caseous tuberculosis of the kidney. Pyelonephritis in the rare cases not originating in a pyelitis may cause blocking of the ureter and pyonephrosis.

Except in the unusual cases of bilateral congenital hydronephrosis which very seldom become purulent, the pus-containing tumour, like hydronephrosis, is generally unilateral.

Symptoms and Signs.—These are, in the main, those already described as belonging to hydronephrosis, and, as in this latter condition, they will vary with the causes which produce the blocking of the ureter. As a rule, there is more pain in pyonephrosis, and fever is always present at some stage of the disease; the hydronephrotic tumour is always larger than the pus-containing one, unless where the latter condition supervenes upon the former. The intermittent discharge of the sac-contents into the bladder will cause a copious pyuria and diminution in the size of the tumour. Usually a pyonephrosis is less movable, owing to the presence of inflammatory adhesions. The urine may be perfectly normal as long as the block is complete in the ureter.

Diagnosis of the Allied Conditions.—The chief difficulty in establishing the diagnosis of pyelitis will be found in the exclusion of *bladder-disease*. This is usually impossible from the examination of the urine alone, which in both cases will contain pus, mucus, some albumin, and occasionally blood, though in pyelitis the reaction is commonly acid and in cystitis alkaline; the amount of albumin is usually greater in pyelitis, especially if tubercular. Little importance must be attached to the nature of the epithelial *debris*, as already stated, since the caudate or club-shaped cells suggestive of pelvic source are sometimes seen in purely bladder cases. Great frequency of micturition is more likely to be of vesical origin, but sometimes it may be of renal source. The presence of a tumour in the renal region, with tenderness and pain, are valuable

diagnostic signs, but the most reliable information is obtained by the cystoscope. By this instrument the orifice of the ureter may be examined and the purulent secretion detected as it flows from the affected kidney into the bladder.

The presence of a renal tumour which disappears or diminishes in size coincidentally with the appearance of much pus in the urine is also pathognomonic.

When renal pus is flowing into the bladder from the kidney, exploratory puncture of the kidney, as resorted to by Senator, may establish the nature of the case; and the injection at the same time of a weak methylene-blue solution through the hypodermic syringe will instantly demonstrate the renal source of the pus—a method useful in the diagnosis of pyelonephritis or abscess of the kidney when the ureter is free.

The differentiation of *calculous* from *strumous* or *tubercular pyelitis* is important. The only reliable test of the latter is the presence of the tubercle bacillus in the urine, the lower urinary tract having been proved free from tubercular disease by cystoscopic examination. H. Fenwick has repeatedly demonstrated the possibility of diagnosing tubercular disease of the kidney in the absence of pain, renal tumour, fever, and bacilli in the urine, by the cystoscopic examination alone, which reveals the characteristic displaced retracted orifice of the affected ureter. The displacement and retraction are the result of thickening of the ureter, which has shortened lengthwise, causing dilatation of its upper and lower extremities, from which may be safely predicted tubercular infiltration and ulceration of these traumatised areas.

The hæmaturia of renal tuberculosis is usually slight, and the attacks of pain caused by the passage of fragments of caseating tissue down the ureter are very rarely, if ever, so marked as the renal colic caused by calculi. The presence of tuberculosis elsewhere, especially of the testicle, and its fever and other clinical symptoms, should be sought for. The Röntgen rays may be employed with advantage, and tuberculin has been found useful also in the differentiation.

Cancer of the kidney rarely leads to pyuria; the hæmaturia is often most copious, and the wasting and cachexia ultimately become extreme.

Perinephritic abscess, if it does not afford a history of injury, will usually be associated with some abdominal condition, as ulceration of the colon, appendicitis, or spinal caries. The previous symptoms of pyelitis or pyelonephritis may be obtained from the history in those cases where the suppuration has spread from the kidney to the surrounding connective tissue. There will be great pain and tenderness, which cause the patient to flex his thigh on the abdomen; the spine is kept rigid, and induration, œdema, and inflammatory thickening become obvious on palpating the loin. The constitutional symptoms are those of high fever, rigors, and sweating, occur-

ring usually in a more acute and severe form than those met with in pyelitis. In the chronic cases the gait is characteristic, the patient walking with his body bent forwards, and the abscess may point at the lower end of the psoas muscle or at Poupart's ligament.

The diagnosis of *hydronephrosis* is detailed on page 665. *Pyonephrosis* is recognised by the history of the cause which has produced it and by the presence of the tumour, which may diminish perceptibly or disappear synchronously with the appearance of much pus in the urine. Bergmann's plan of washing out the bladder and, whilst the catheter is still *in situ*, squeezing gently the renal tumour, is a valuable diagnostic method; when the ureter is not blocked the pus may be made to flow through the instrument.

Acute pyelitis of childhood, due to invasion by the bacillus coli, is recognised by the highly acid urine which is swarming with the bacilli, and the condition, as first pointed out by Holt, is rapidly relieved by alkalies.

Prognosis.—The outlook in pyelitis depends upon the cause; a favourable termination may be constantly expected in those cases where the affection is the result of irritant drugs or of the toxins and bacilli present in the exanthemata. In calculous pyelitis the passage down the ureter of the irritating concretion may occasionally lead to the disappearance of the symptoms, and the same result may follow upon the passage of hydatids or other parasites. Where the causes are permanent, and especially when pyonephrosis has already become established, the prognosis becomes serious, and should symptoms of pyelonephritis supervene it assumes usually a grave aspect. Even in cases where the ureter becomes permanently blocked, life may be prolonged for many years; and when the duct remains pervious large quantities of pus may continue to be passed in the urine for an indefinite period without serious deterioration of health.

In the tubercular form a fatal issue may be confidently predicted, though the duration of life may extend to five or seven years in some cases; many museum-specimens demonstrate the complete drying-up of the caseous material and its replacement by a mass of calcified matter, death having occurred from some other affection.

The advent of amyloid disease in the opposite kidney or the constant presence of ammoniacal urine in the bladder is invariably a grave symptom.

The most important element in prognosis will always be that which operative treatment introduces, and the hope of success largely depends upon the integrity or adequacy of the opposite kidney.

Treatment.—In the acute cases caused by the irritation of drugs or toxins little is needed beyond rest and diluents, with the use of hot baths and mild diuretics like citrate of potash.

In the chronic cases with much pyuria antiseptic drugs like boric acid, urotropin, salicylates, carbolic acid, &c., may be administered. The writer has found the most decided benefit from the administration of creosote, in the purest form, given in doses of 3 mins. in a capsule three or four times a day.

Albarran has recorded many successes by Casper's method of catheterising the ureter and flushing out the renal pelvis with a dilute perchloride of mercury or a strong solution of boric acid. The washing-out of the bladder is much less useful than the sterilisation of the urine by antiseptics administered by the mouth. The prevention of pyelitis in retention of urine is a most important matter; the writer can recall several cases where the condition followed the use of septic catheters and one fatal case following the introduction of a bladder-sound which had not been sterilised.

The main hope in the treatment of chronic pyelitis must always lie in the removal of the cause; the prevention and cure of the condition has been greatly assisted by the new operation for the removal of the prostate gland.

The surgical procedure for the relief of pyelitis is to cut down upon the kidney, examine its condition, and by nephrotomy, after exploring its contents, freely to remove all diseased tissue, pus, or concretions and provide for suitable drainage. Where the organ is found destroyed, as in pyelonephritis and pyonephrosis, the operation of nephrectomy, or removal of the entire kidney, should be performed. In calculous pyelitis and in the chronic tubercular form the results are often most satisfactory if operation be not too long delayed.

It is of vital importance in all operative cases to determine the integrity of the opposite kidney and the degree of value to be attached to its secretory power before the performance of any radical operation. The catheterisation of the ureter and a careful examination of the urine collected will afford valuable information. The value of determining the freezing-point of the blood has been already referred to. Phloridzin administered hypodermically affords an indication of the adequacy of the organ by noting the amount of sugar excreted in the urine soon afterwards. When it has been rendered certain that the function of one kidney is entirely destroyed by the permanent blocking of its ureter, the determination of the renal adequacy is a simpler matter, and Wright's method affords convenient and reliable results. This plan also should be resorted to in prostatic or bladder cases where the operation of nephrectomy is not under consideration, but where the operative procedure is about to be undertaken for the relief of the disease in the lower urinary tract.

PYELONEPHRITIS and PYONEPHROSIS

Are described in the preceding article.

PYLEPHLEBITIS. (See under THROMBOSIS and LIVER.)

PYLORUS

Diseases affecting the pyloric opening are described under 'Gastric Ulcer' and 'Gastric Cancer.'

PYOPNEUMOTHORAX.

(See under EMPYEMA and PNEUMOTHORAX.)

PYURIA

Pus in the urine is but a sign of many diseased processes affecting the genito-urinary tract; it may also be the result of an abscess which has perforated into the urethra, bladder, ureter, or renal pelvis. These latter extraneous or extrinsic sources of pyuria most commonly are met with in women suffering from disease in the pelvic organs. Thus it is not uncommon for the pus resulting from the breaking-down of a pelvic hæmatocele or cellulitis to find its way into the bladder. Pyosalpinx, abscesses of the ovary, and occasionally suppurating ovarian or dermoid cysts may discharge into the bladder and cause pyuria. Purulent secretions coming from the uterus and vagina blend with the bladder-contents as these are being expelled in the act of micturition, and a similar contamination occurs in the bladder when a vesico-vaginal fistula exists. In either sex psoas-abscesses, sacral or hip-joint disease, and perityphlitic mischief may be the source of the pus discharged in the urine. In all these cases there will usually be in the history of the case evidence afforded of the primary suppurative condition. Often the purulent discharge bursts into the urinary tract suddenly, so that enormous quantities appear in the urine for a day or two, after which only faint traces may be discernible. The writer has once seen many pints of pus evacuated in the urine in a single night from the rupture of an old vertebral abscess. When the pus enters the bladder from the perforation of an abscess in connection with the bowel, gas may accompany it, the condition known as *Pneumaturia* being produced, in which

gas may be passed with an audible sound towards the end of micturition. Occasionally, however, pneumaturia may exist without obvious pus, in which case it has been supposed to be the result of gas-forming microbes accidentally introduced, and in glycosuria the yeast-fungus may lead to the production of small quantities of carbonic-acid gas. In all these cases the pus is free from renal elements, unless old-standing irritation induces secondary ascending renal changes.

When the pyuria is the result of urethral inflammation, as in gonorrhœa, its spontaneous discharge independent of micturition is quite characteristic, and gonorrhœa-threads in the urine are also obvious. In old or long-standing gleet the urine passed into a vessel at the commencement of the act of emptying the bladder will be found to contain pus, whilst that flowing afterwards is quite free once the urethra has been flushed out. In prostatic or posterior urethral cases pressure upon the perinæum or through the rectum will cause a discharge to exude from the urethra towards the end of micturition. The urethroscope will reveal ulcerations of the canal which discharge small quantities of pus when the ordinary signs of urethral inflammation are absent.

The pus coming from vaginal discharges is absent from the urine when careful sponging is carried out before the patient passes water ; but when doubt exists, the absence of pus in a catheter-obtained specimen is conclusive.

Cystitis is a frequent source of pyuria ; the pus escapes with the urine, with which it may be intimately mixed ; there are signs of bladder-inflammation—increased frequency, pain, &c. Upon standing, the specimen may show a considerable amount of pus and mucus, which fall as a deposit as long as the urine is acid ; but in long-established cases the pus is more intimately blended, remaining in suspension with the mucus and forming a turbid glairy mixture, which is alkaline and often foul-smelling, and contains micro-organisms, crystals of triple phosphates, bladder-epithelium, and blood. Towards the end of micturition in these cases there may be usually observed a discharge of thick ropy pus or mucopurulent matter ; decomposition of the secretion is already far advanced before the urine is voided, and there are swarms of proteus bacilli, staphylococci, &c., in the highly alkaline fetid liquid.

As a rule, the pus found in alkaline urine is of vesical origin, whilst that existing in acid urine probably comes from the kidney. Many mistakes will be made, however, if this rule be accepted without serious reservations. Thus in mild vesical catarrh the reaction is usually acid, especially in women, and for a long time also in males. The alkalinity or acidity is largely determined by the influence of the prevailing microbe : the bacillus coli and the tubercle bacillus produce cystitis

with acid urine, whilst the previously mentioned organisms, by splitting up or decomposing the urea, cause a high degree of alkalinity, and this is especially true of *B. proteus*.

It must always be remembered that in pyelitis associated with cystitis the reaction of the urine is frequently alkaline.

Before the diagnosis of a purely vesical origin of the pus can be demonstrated the cystoscope should be resorted to. In bladder-disease the signs will be apparent; if the pus is coming from the kidney, catheterisation of the ureters will demonstrate which organ is at fault, and it will afford means of investigating the urine coming direct from the kidney; De Luy's segregator is less reliable.

When the pus has been proved to be neither of extraneous source nor derived from the urethra, prostate, or bladder, the next step in the investigation is to determine what particular form of renal disease is present causing the pyuria.

The differential diagnosis will lie between pyelitis (calculous or tubercular), pyelonephritis, acute suppurative nephritis, and pyonephrosis. The condition of the urine in each of the above affections is discussed in detail in the article upon 'Pyelitis,' where the symptoms and signs of these allied renal affections are described, and need not be here repeated. Cystic disease of the kidney in which the cysts have suppurated is not, however, discussed under 'Pyelitis.' The condition is extremely rare; and when pyuria has supervened the history is obtainable of bilateral renal tumours of large dimensions, associated with recurrent hæmaturia and the signs, symptoms, and characteristic urine of a chronic interstitial nephritis. Amongst the latter are to be noted copious low s.g. urine, with hyaline or coarse granular casts and albuminuria, cardiac hypertrophy, arterio-sclerosis, and sometimes bronzing of the skin.

RABIES. (See under HYDROPHOBIA, p. 667.)

RAYNAUD'S DISEASE

This rare affection, also known as Symmetrical Gangrene, Local Syncope, and Local Asphyxia, manifests itself by an arrest of the arterial supply to some part of the peripheral circulation, which causes a local syncope, followed by signs of local asphyxia or cyanosis, and often by symmetrical gangrene of the affected tissues. The local syncope, asphyxia, and gangrene were regarded by Raynaud as successive stages in the attacks, but, as will be seen, sometimes the seizure never extends beyond the first stage; sometimes the first stage is absent, and even the gangrenous stage has been known to

suddenly appear without the usual premonitory syncope and asphyxia.

Etiology.—Though no age is exempt, the disease is rare at the extremes of life, being, in the great majority of recorded cases, observed in young adults. Girls are more frequently affected than boys, and young men more than young women; but upon the whole the female sex suffers much more frequently than the male. A neurotic history is found in many cases, and sometimes hysterical manifestations are present, but a direct hereditary history of the affection is certainly rare. Many of the subjects of Raynaud's disease complain of cold hands and feet, and a history is obtainable of 'dead' fingers or toes, in which the digits, after exposure to cold, become numb and waxy-looking.

The most frequently found exciting cause is exposure to cold; hence the disease is generally observed in winter, and especially after the extremities have been chilled by cold water. Sometimes a history of severe mental or muscular exhaustion immediately preceding the exposure may be obtainable. In other cases the disease has been preceded by an attack of measles, scarlatina, typhoid fever, malaria, or syphilis, and it has been recorded as occurring during Graves' disease, diabetes, and often in association with hæmoglobinuria and various skin-diseases, especially generalised sclerodermia and urticaria. In a considerable number of cases arthritic mischief is associated with the affection, as seen in ankylosis of the end-joints, and in a few cases mental or cerebral symptoms, and sometimes diphtheritic multiple neuritis and transient eye-symptoms, have been recorded.

Pathology.—Of this nothing is definitely known, though the original assumption of Raynaud that the disease was the result of spasm in the arterioles is probably correct. The arrest of the arterial blood-supply affords an explanation of the local syncope, and the resulting venous stasis and capillary dilatation, followed by transudation of the blood-elements into the tissues in the immediate neighbourhood of the obstruction, would explain the subsequent phenomena. Whether the spasm is of centric origin as thought by Raynaud and Buzzard, or the result of peripheral nerve-irritation as maintained by others, remains to be proven. The tendency of recent observers, including Pitres and Crocker, is in the direction of accepting as the most likely explanation of the vaso-motor disturbance a toxic origin of the disease—a view supported by the association of multiple neuritis and hæmoglobinuria with the disease.

Symptoms.—The fingers and toes, usually after some exposure to cold and damp, become hard, waxy-looking, and cold. Generally the attack is confined to one or two fingers, the thumb usually escaping, but occasionally the whole hand

may be involved ; and not rarely the nose and ears, and even the tip of the tongue, also suffer. The pallor is associated with numbness, and is followed after a variable period (sometimes within less than an hour) by swelling and smarting or stabbing and burning pains. The affected digits become hot, congested, and livid, and within a few hours to several days the colour may approach to deep blackness ; this constitutes the stage of local asphyxia. It may occur without local syncope, and more rarely the syncopal phenomenon may pass off without being followed by the asphyxial signs ; as many as a score of such attacks have been recorded in a single day, passing off sometimes as suddenly as they came. Local sweating sometimes occurs, and occasionally desquamation follows, and the nails may be shed. Such attacks may continue to recur at intervals for years, and often some of them have not been preceded by exposure to any kind of cold, the exciting cause being apparently mental or due to some transient illness. The attack may be confined to the hands or to the feet, or to all four extremities ; rarely is it unilateral. Sometimes there is little or no pain, but anæsthesia may be marked in the affected digits of one hand while severe burning pain may be experienced in the affected fingers of the opposite side.

The stage of asphyxia may gradually pass off, but frequently gangrene occurs, though only in rare cases is this latter severe or extensive. The gangrene is as a rule dry ; only occasionally do numerous bullæ form. In mild examples of the disease the finger-tips become the seat of small necrotic areas, involving only the superficial layer of the skin. Often after a series of mild attacks the ends of the fingers may exhibit numerous scars ; but generally, as these slighter seizures occur, they show a tendency to take on a more severe character. The sloughs become deeper, a line of demarcation forms, and the gangrenous part is slowly separated ; the phalanges are shed, usually without undergoing necrosis, leaving a slowly healing conical stump. The distribution of the gangrene varies considerably ; it is seldom typically symmetrical ; thus one digit may become gangrenous whilst one of its neighbours may remain cyanosed or asphyxiated, and the adjoining one may not pass beyond the stage of pallor. As a rule, there is involvement of the corresponding digits upon the opposite side, though one or more finger-tips on one hand may become gangrenous whilst the opposite corresponding digits may escape after passing through the asphyxial stage.

In children the gangrenous process may be sometimes very severe and rapid, necessitating amputation of a considerable portion of one or more limbs. Rarely does death of any but a peripheral part occur, but occasionally the calf and even portions of the trunk may necrose. The ears, nose,

genitals, and eyelids may suffer, but usually only to a slight extent; the majority of patients escape with little more than the loss of a portion of the pad of one or more fingers or toes.

In rare cases the type of the disease assumes a chronic character, the local asphyxia being more or less continuous instead of paroxysmal, and more rarely still symmetrical gangrene may appear without being preceded by local syncope or asphyxia.

Whilst the most obvious feature of the disease consists in its frequently recurring paroxysms, cases are sometimes observed which, after one severe attack, ending in resolution or gangrene, never show a recurrence. The frequency with which the disease is associated with paroxysmal hæmoglobinuria is noteworthy. Sometimes the hæmoglobinuria has been found to precede the disease, at other times it appears as a secondary complication, whilst rarely it occurs between the attacks, apparently after exposure to cold, taking the place of a paroxysm of the disease. Albuminuria and epistaxis are occasionally observed.

Diagnosis.—*Diabetic gangrene* is differentiated by the history of the case and the detection of sugar in the urine; the gangrene is often of the moist type, and there is evidence of multiple neuritis.

Senile gangrene occurs in old subjects, is very rarely if ever symmetrical, the hands are not involved; usually the entire foot suffers, and disease in the arterial walls can be detected, in marked contrast to the recurrent paroxysms of Raynaud's disease, in which hands and feet suffer, but only with the partial loss of superficial and distal portions.

Ergot-poisoning may simulate the disease in its first attack, but in the history of ergotism the epidemic character is distinctive.

ERYTHROMELALGIA.—This resembles Raynaud's disease. It is a rare affection described under the title of Erythromelalgia or Red Neuralgia, in which the main symptoms, as first described by Weir Mitchell, consist of severe paroxysmal pains in the extremities, commonly in the lower ones, associated with heat, great redness, and swelling. The attacks, like those of Raynaud's disease, occur with great frequency, often being brought on by exposure to cold or by the fatigue of standing. All the symptoms are much aggravated by a dependent position of the limb, by any muscular exertion, and by warmth. The pain may, in rare cases, precede the redness for several days, and, as in Raynaud's disease, the deep redness and swelling may occur before the symptoms usually heralding an attack.

A paroxysm of agonising throbbing pain commonly begins in one foot, and within some minutes or hours a bright redness, starting in patches, covers the entire foot; the skin becomes

swollen, and if the limb be held in the dependent position the skin may pit on pressure, the arteries throb, and the veins dilate. After an hour or two, sometimes much sooner, the bright redness gives way to a discoloration like that observed in Raynaud's disease, in which the foot may become deeply cyanosed, and the pain, which has grown in intensity during the red stage, gradually fades away, unless the foot be put to the ground. In an attack, walking is usually impossible owing to the agony caused by movement and by the dependent position. The local heat in the affected part subsides with the onset of local asphyxia, and relief is usually obtainable by the application of cold water when the erythematous redness is at its height. As little is known of the pathology of the affection as is known of the pathology of Raynaud's disease, but the vaso-motor origin of the attacks is obvious. The site of the irritation is supposed to be in the cornua of the cord, or in the medulla, since organic spinal lesions have sometimes been known to supervene. Weir Mitchell regarded the affection as the result of a peripheral neuritis, or of an interruption of the sensory fibres, but marked muscular wasting and anæsthesia do not occur. Children are seldom affected: the majority of the recorded cases have been seen in males, about middle-age.

That the affection is closely related to Raynaud's disease is evident from the symptoms and from the fact that both conditions have been observed in the same subject. The attacks are, however, not symmetrical; though the feet are usually affected, one foot only is generally involved in the seizure, the other suffering in a later attack. Sometimes the hands and arms suffer, but not so frequently as the feet and legs; the lumbar region and the nape of the neck have been seen, in a few cases, to be the site of the pain, redness, turgescence, and lividity. Trophic changes are sometimes observed after repeated attacks, the skin becoming glossy and the subcutaneous tissues showing atrophy. Local sweating often occurs during a severe seizure.

Diagnosis.—Both erythromelalgia and Raynaud's disease are differentiated from *syringomyelia* by the paroxysmal nature of the attacks, by the absence of the characteristic loss of sensation to pain and to variations of temperature combined with muscular atrophy. In Morvan's type of *syringomyelia* the trophic changes may possibly be mistaken for those seen in Raynaud's disease, but they are for the most part painless, resembling those seen in locomotor ataxia, and, as pointed out by Purves Stewart, necrosis of bone and painless whitlows are rare in Raynaud's disease. *Frost-bites* are seldom so symmetrically distributed as the local syncope of Raynaud's disease; there is always a history of recent exposure to intense cold. *Chilblains* can be only mistaken for either disease

when unusually severe, but they always lack the characteristic paroxysmal feature.

Prognosis.—In Raynaud's disease the outlook only becomes grave in the rare cases occurring in childhood, in which multiple gangrene may terminate life within a few days. Even in the milder chronic cases the general health is usually unaffected, and as long as gangrene does not occur there is little to be feared. Most cases never eventuate in the death of any considerable amount of tissue, and after years of recurrences the frequency of the seizures gradually diminishes. In erythromelalgia a fatal result never occurs save from some spinal development, as disseminated sclerosis, but the disease usually pursues an indefinitely protracted course; very occasionally only do the attacks cease to recur.

Treatment.—The object should be to protect the individual from changes of temperature, and those who can should spend their winters in a warm equable climate. The underlying conditions, as syphilis, malaria, renal disease, &c., should be met by appropriate treatment. For the arterial spasm galvanism has been highly praised when used upon Barlow's plan of immersing the limb in a tepid salt solution, in which the positive pole of the battery is placed, the negative being applied to the limb, above the water-level.

Gentle massage between the attacks, combined with Swedish movements, is indicated. Gangrene when it occurs must be treated upon recognised surgical principles.

The treatment of Erythromelalgia is still less satisfactory; the chief remedies to be relied upon are rest in the horizontal position, elevation of the limb, and the application of cold during the attack.

RELAPSING FEVER

This, also known as Famine Fever, Spirillum Fever, Febris Recurrens, and Typhus Recurrens, is an acute infectious disease caused by the presence in the blood of a spirillum which produces a severe fever, of abrupt onset, lasting for about six days, to be followed by an apyrexial period of similar length, after which a second invasion occurs, usually of somewhat shorter duration.

Distribution.—The disease visited Ireland several times during the last century, and it is stated by some authorities to be endemic in the island; but the writer has never seen a case of it, and this is also the experience of Sir John Moore. Though a few deaths are still usually registered annually in Ireland as due to relapsing fever, the last-named authority considers it probable that these are examples of some other affections, since the disease is distinctly contagious. It is

difficult to conceive that an affection showing epidemic characteristics could be constantly lurking about and only manifesting its presence by one or two fatal cases during each year. It seems to be endemic in some parts of India and Russia, and epidemics have been recorded at times over different parts of Europe, Asia, Africa, and America.

Etiology.—As implied in its name (Famine Fever), the disease, like typhus, appears in conditions where destitution, scarcity of food, and overcrowding, with bad ventilation, prevail. Both affections have generally been observed in the same epidemic. It is uninfluenced by season, climate, or soil, attacking both sexes and all ages and all races. It is markedly contagious, and one attack does not confer immunity. The disease has been conveyed to men and monkeys by inoculating them with the blood of patients suffering from the malady.

The specific cause was demonstrated by Obermeier to be a slender flagellated spirillum, spirochæte, or protozoan organism measuring from 20–40 μ . This active motile filament is seen darting about energetically in the fresh blood drawn during the pyrexial periods, but it has not been detected in the secretions. Metschnikoff found it in the spleen after the subsidence of the fever. The blood contains during the apyrexial period refractile bodies resembling diplococci; these appear also in great numbers immediately before the onset of the second attack of fever. They are regarded as spores, and von Jaksch believed that he saw these organisms assume the form of short thick rods, which afterwards developed into the slender spiral filaments constituting the perfect form of the *spirochæta Obermeieri*. These specific organisms or their spores are probably conveyed from the diseased to the healthy by fomites, or in the exhalations given off from the body, and it has been demonstrated that the spirilla or their spores can be inoculated by the bites of bugs and other parasites. The organism has not as yet been cultivated outside the blood in artificial media; nothing is known about its life-history; it is highly sensitive to temperature above the range of fever-heat, and seems to be destroyed readily by the addition of water to the blood containing it.

Symptoms.—After an incubation-period, which is said to range from a few hours to a week or more, the invasion occurs with abruptness, the disease being ushered in usually by a smart rigor, backache, headache, muscular pains in the limbs, and vertigo. In children convulsions are common. Rarely are prodromal symptoms observed. After the initial chill the temperature rises suddenly, and may register 105° or 106° within twelve or twenty-four hours, and the pulse becomes markedly accelerated, much beyond that observed in other pyrexial conditions. The usual concomitants of severe fever are present. The tongue, however, usually remains moist,

though covered with a thick yellowish-white fur; there are thirst, constipation, nausea, and anorexia, and sometimes severe vomiting, especially in children; but occasionally a voracious appetite is seen in some epidemics. The vomit is often green, and sometimes coffee-ground in character; as a rule, intestinal symptoms are absent, the only abdominal signs being marked splenic enlargement, and often distinct jaundice, with swelling of the liver. The jaundice is usually slight, but in the malignant type of the disease, described by Griesinger as *bilious typhoid*, the icterus is intense, and is associated with cyanosis of the nose, coldness of peripheral parts of the body, and severe colic.

The fever remains high for five, six, or seven days in severe cases, and just before defervescence a temperature of 108° may be registered; the crisis is abrupt, the temperature often falling 10° F. and the pulse registering half the number of beats recorded twelve hours previously. With the sudden fall in the fever-heat copious drenching acid perspirations are observed, and occasionally smart diarrhœa and epistaxis. Delirium is present when the fever is high; upon the whole it is less marked throughout than in typhus, but often wild delirious symptoms appear shortly before the crisis. In a few days the patient's temperature and pulse rise from the sub-normal to the normal, and remain so; he feels quite well, and can usually be restrained with difficulty from moving about in this apyrexial period.

After about the seventh day from the crisis—the fourteenth from the initial chill—the characteristic relapse occurs. This is marked by the same abrupt invasion, with rigors, followed by high temperature, and the other symptoms of severe pyrexia. Though the fever may be more severe than in the first attack it is usually of shorter duration, the second sudden crisis usually occurring after the third or fourth day, with profuse perspiration. In some cases a second or even a third relapse has been observed; more rarely the primary attack of fever is not followed by any relapse, convalescence being uninterrupted.

There is no specific rash, but sudamina commonly develop during the sweating stage; occasionally herpes appears on the lips, and in rare cases a roseolar eruption has been observed, and even petechiæ may occur.

Complications are not very commonly present, but pneumonia is not rare; hæmorrhage from the nose, stomach, or bowel occasionally occurs, even to the extent of producing serious or fatal collapse. Nephritis is perhaps the most common of all complications, if jaundice be regarded as a symptom of the disease.

Rupture of the spleen, or splenic thrombosis, may set up a fatal peritonitis, and dysenteric complications have been recorded in some epidemics. Amaurosis has frequently

been witnessed in many epidemics as a sequela of the disease : the mischief apparently results from an inflammatory action, first starting in the choroid and ciliary body, and it has been stated by Murchison to sometimes end in permanent loss of vision ; occasionally ophthalmia has been observed. Laryngeal oedema occasionally complicates the disease, and cough is common, from bronchial irritation or pharyngitis.

Diagnosis.—The easy demonstration of the *spirochæta Obermeieri* in the blood during the pyrexial period settles the diagnosis at once. In the early stage of an epidemic *typhus* will be naturally suspected ; and it must always be borne in mind that, though most epidemics are of a mixed nature, the tendency is, towards the end, for typhus to take the place of the relapsing fever. The characteristic rash and fourteen days' duration of typhus are distinctive.

Prognosis.—The general mortality of the disease does not exceed about 4 per cent., and it is highest in old subjects, especially in those whose hearts are the seat of structural disease. Pregnancy renders the prognosis grave, abortion being the rule, and often death follows.

Treatment.—No reliance can be placed upon any drug for the reduction of the pyrexia or the prevention of the relapse. The disease must be treated by rest in bed, skilled nursing, fluid dietary, and the discreet use of stimulants after the commencement of the precipitous crisis. Symptoms as they arise should be treated upon approved therapeutic principles. The prophylaxis of the affection is obvious, since destitution, poverty, dirt, and overcrowding are the constantly present factors in its origin and dissemination.

RENAL DISEASE.

{Under BRIGHT'S DISEASE, KIDNEY, PYELITIS, &c., the different affections are described.}

RETROPHARYNGEAL ABSCESS.

(See under PHARYNGITIS, p. 1289.)

RHEUMATIC ARTHRITIS.

(See under ARTHRITIS DEFORMANS, p. 69.)

RHEUMATISM, ACUTE

This is an acute fever, caused by the presence of a specific micrococcus, which produces characteristic sweating and transient multiple arthritis, associated with a marked tendency towards pericardial and endocardial inflammation often terminating in permanent valvular deformities.

Etiology.—Though the disease has a wide distribution, and is met with in all climates, it may be regarded as an affection most commonly prevalent in cold and damp cities. Season has a marked influence, the disease being much more frequent in spring and autumn, especially in cold, humid, autumnal seasons which follow dry, hot summers, when, according to Newsholme, there is a low level of the subsoil water. Kleinschmidt states that the incidence of the disease, as proved by the mortality returns, varies directly with the amount of rain or snow which falls. Sex-influence is seen in the slight preponderance of males in all the large collections of cases. All ages supply considerable numbers, but the disease is decidedly more frequent in late childhood and young adult life, and becomes rare after fifty. As a rule, the heart-manifestations are more severe in children than in adults.

Hereditary predisposition or vulnerability cannot be denied, and many authorities regard a considerable proportion of the examples of congenital valvular disease as the result of an attack of the disease during foetal life. One attack does not confer immunity, but upon the contrary renders the patient more liable to subsequent seizures. Exposure to cold and damp acts either as an exciting or predisposing cause, a history of recent chills or wettings being obtainable in a variable but considerable percentage of cases, often such history being associated with previous mental or physical over-strain. Occupation operates as an etiological factor in proportion to the facilities which it affords for sudden exposures to cold and wet after the skin of the body has been perspiring freely, or in proportion to the frequency with which young outdoor labourers are subjected to prolonged wettings.

Like pneumonic fever, acute rheumatism has been occasionally observed to occur in epidemic form. This latter fact, taken in conjunction with evidence that the disease has been known to attack several inmates of particular houses, and that its complications and many of the phenomena of the disease itself resemble those observed in septic affections, has long led many observers to regard acute rheumatism as an infective disease.

The subject is, however, still surrounded with great difficulties, not the least of which is that many different micro-organisms have been demonstrated in the blood, joints, and

affected tissues. These have been usually streptococci and other pyogenic cocci, probably the result of multiple infection, and cannot be regarded as evidence of the specific causation of a disease universally recognised as showing little or no tendency towards the formation of purulent collections.

The researches of Poynton and Paine, already mentioned in the article on Endocarditis, deserve the most serious consideration. These observers claim that, after many years of investigation, they have been able to isolate the specific micro-organism of rheumatic fever from the blood, urine, and local lesions; they consider it is identical with the organism discovered by Tribolet and by Wassermann. It is a minute micrococcus appearing in pairs; each element of the diplococcus measures 0.5μ in diameter. In solid media it grows in staphylococcal masses, whilst in bouillon it forms streptococcal chains consisting of diplococci. This organism, now known as the *micrococcus rheumaticus*, so closely resembles streptococcus pyogenes that several observers still maintain that it is but a variation of the latter. The recent researches of Beattie, however, have placed the matter practically beyond doubt; he finds that the micrococcus rheumaticus differs completely in most of its cultural peculiarities from streptococci, and that the results of its inoculation in animals are also entirely different. His conclusions agree with those of Poynton and Paine, and appear to absolutely justify the contention that the micrococcus is the specific cause of acute rheumatism. Thus he finds that it can be isolated from typical acute cases of the disease, can be cultivated outside the body, and when inoculated into animals produces a replica of the picture of human rheumatic fever, affecting the joints, endo- or pericardium, sometimes causing chorea; and not seldom all these phenomena are produced in the same animal. Even more important still is the demonstration that from the animal lesions the micrococcus can be isolated in pure culture. Poynton, as early as 1904, showed that the micrococcus during the progress of the rheumatic fever grows best in the local lesions, and is found with difficulty in the blood, joint, or pericardial exudations; but it may be discovered after death lying deeply in the synovial membrane, in the pericardial tissue, and in the vegetations present on the cardiac valves. Dr. V. Shaw was able to produce a fatal arthritis, pericarditis, and endocarditis in monkeys by injections of a culture of the micrococcus obtained by Poynton from a typical case of rheumatic fever associated with chorea and valve-disease in a girl. The identification of the specific micrococcus, and its essential difference from the ordinary streptococci, have been also demonstrated by Beaton and Walker, who proposed the name '*micrococcus rheumaticus*' for the organism to distinguish it from the streptococci which

are sometimes found in cases of rheumatic fever showing multiple infection. The results of these bacteriological investigations can be reasonably claimed to be decisive, and the theories of a nervous, lactic acid, or metabolic origin of the disease may be safely discarded. In the same way the theory may be disposed of that acute rheumatism is of pyæmic nature. The presence of the pyogenic cocci, occasionally found in the disease, is obviously accidental, the tendency of the rheumatic inflammation being entirely different from that produced by streptococci when they invade the human body, as extremely seldom do the joints suppurate. Such cocci, when introduced into animals, always cause a suppurative arthritis, if the joints become invaded, whilst the micrococcus rheumaticus produces a non-purulent arthritis, apparently in every respect identical with that observed in the human affection. The conception of the infective nature of the disease and of its being the result of the invasion by one specific micrococcal organism in no way clashes with the operation of other etiological factors, as season, climate, subsoil, water, exposure to cold, &c., a parallel case being seen in pneumonia, which undoubtedly often speedily follows exposure, and has a marked seasonal preference. Probably the micrococcus rheumaticus will ultimately be found to be a constant resident in the bodies of healthy subjects. Newsholme predicted that it would be found to be a saprophyte spending part of its existence in the soil and part as a parasite in man. Poynton and Paine satisfied themselves that the organism gains admission to the blood through the tonsils. The writer many years ago suspected the infective nature of the rheumatic poison by witnessing an outbreak of the disease in horses confined in a particular stable, before bacterial investigation was feasible. The identity of the disease with human acute rheumatism was rendered highly probable, if not demonstrated, by the remarkable therapeutical action of salicylate of soda, which always promptly for a time removed pain, fever, joint-swelling, and lameness.

Morbid Anatomy.—The joints in fatal cases exhibit the usual changes observed after severe acute inflammation. There is congestion of the synovial membrane with effusion within and around the joint, the fibrous and ligamentous tissues being swollen and infiltrated with leucocytes. As already stated, the specific micrococcus may be absent from the joint-exudation, but it is found in the synovial membrane in the areolar tissue lying beneath the endothelial lining.

The changes generally accepted as characteristic of inflammation of the endocardium and pericardium are usually present, and the organisms are recognisable in the nodules or vegetations on the cardiac valves, especially upon those of the left side of the heart. The myocardium in severe long-standing

cases is the seat of fatty degeneration and sometimes of advanced fibrosis. The pleura is often affected, the spleen commonly is enlarged, sometimes advanced cloudy swelling and granular or fatty changes are observed in the kidneys, and the lungs may be the seat of pneumonic changes. The blood contains an excess of fibrin, and after death firm coagulation is usually apparent. Very rarely are there any evidences of pus in the joints or in other regions, but infarcts are observed in cases where the endocarditis is of the malignant type.

Symptoms.—The onset of the disease is as a rule abrupt, but never strikingly sudden, as there is very seldom an initial distinct rigor, even when the affection has appeared in its acutest form after a recent exposure. In a small percentage of cases there are prodromal symptoms, of which the most important is sore-throat, coupled with lassitude and malaise. Very often the first intimation which the patient receives of illness is smart pain or stiffness in a joint; with this is often experienced slight chilly sensations. In a few hours or more intimations of further joint-troubles are received, and within a day or two several articulations are involved. The first joint seized upon is often the largest in the body—*i.e.* the knee. The subsequent extensions vary considerably: often the corresponding joints suffer consecutively; thus the mischief in the knee is followed by similar trouble in the elbow, the wrists and the ankles suffer together, the shoulder-pains fly to the hip, and the joints of the hands and feet often suffer simultaneously. There is soon established an unmistakable multiple arthritis of very fugitive type, one joint clearing up as others become involved. Upon inspection the joints exhibit the usual clinical manifestations of inflammation: they are hot, red, and swollen, but the intra-articular effusion is very rarely extensive. They are the seat of severe pain and are usually exquisitely tender to pressure. Upon examination the swelling can be made out as existing not only within the joint, but in all the tissues surrounding it. This is well seen in the wrists and fingers, where the tendinous sheaths and fibrous structures are very obviously swollen, causing more or less uniform enlargement of the entire hand. Very rarely, if ever, is the attack confined to a single large joint, but mild seizures may only involve the hands and feet or the knees and elbows. It is not rare, however, to find only a single joint affected at a given time; thus for a day or two the wrist may suffer alone, and, as the local symptoms pass off, the ankle upon the same or upon the opposite side becomes involved; more frequently, however, both ankles and both wrists suffer together.

The vertebral, sacro-iliac, sterno-clavicular, and temporo-maxillary articulations do not always escape, but their invasion

is so rare as to raise a doubt, when it does occur, that the disease is acute rheumatism.

With the joint-manifestations the usual symptoms of acute fever are present: the temperature rises rapidly, reaching often to 102° or more by the end of the first twenty-four hours. The range is irregular in type and pursues no definite course, fluctuating considerably with the implication of fresh articulations and fading transiently as the pain, swelling, and redness pass away. The only characteristic feature in the temperature of acute rheumatism is its tendency to rush up to hyperpyrexial heights without obvious warning; but this complication only occurs, fortunately, in a small proportion of cases. The prominence of the pyrexial symptoms explains the origin of its popular synonym—Rheumatic Fever.

Sweating is constantly present; usually it is more or less profuse and often drenching; the secretion is always marked by an unmistakable sour odour, even when not reddening litmus-paper. With the perspiration are commonly associated copious sudaminal or vesicular eruptions in which more or less erythematous redness may surround each vesicle. These are not to be confused, however, with the other cutaneous complications to be presently mentioned. In connection with the characteristics of the perspiration it is interesting to note that Walker and Ryffel found that the micrococcus produces considerable amounts of formic and other acids of the fatty series. Appetite fails, the tongue is coated with a thick white fur, thirst is often severely complained of, constipation is the rule, the urine is scanty, strongly acid, and high-coloured, and deposits abundant urates, but the chlorides are diminished. Delirium is rare, unless in high pyrexia, the mind remaining usually very clear and alert throughout. The pulse early becomes rapid and soft, so that it is easily compressed, but, unless cardiac involvement occurs, it is regular. The heart, even at an early stage, often exhibits some signs of dilatation, the area of cardiac dulness being increased, the apex-beat diffused, and the sounds approach each other in character. With the advent of acute peri- or endocardial mischief other signs appear. The blood deteriorates rapidly, anæmia becoming well marked often within a week, and usually there is well-marked leucocytosis.

The duration of acute rheumatism varies greatly. Before the routine administration of salicylates it was common to observe patients suffer for many weeks with little or no relief to their distressing pains, sweats, and fever. Owing to the prompt relief afforded by these drugs to all the symptoms, it is almost impossible to form any accurate conception of when the attack has passed off, unless by suspending their administration. The presence of heart-mischief, pleural, pneumonic, or other complications may prolong the duration

of the attack for a very considerable number of weeks. Owing to the anæmia and debility left after the disappearance of the fever, and because of the stiffness of the joints remaining after the fever has vanished, the convalescence is usually slow and protracted, and relapses are often observed.

Complications.—The advent of cardiac mischief during an attack of acute rheumatism should be regarded not as a complication, in the strict sense of the term, but as a manifestation of the disease, like the arthritic phenomena. Indeed, in the case of very young subjects there are good grounds for believing that the disease often manifests itself by the influence of the toxins upon the heart, the joints escaping. The first signs of cardiac affection are usually the evidences of dilatation already referred to, the poison depressing the tone of the myocardium without causing the usual signs of pericardial or endocardial disease; these may be assumed, however, to be present also, though probably in a lesser degree. This *carditis*, as it is called, may be accepted as very frequent; probably it always exists, except in the mildest cases. When the main action of the poison is expended upon the lining membrane, endocarditis is said to be present; and when the fibrous covering of the heart is chiefly involved, pericarditis becomes demonstrated. The symptoms and signs of these conditions are described under their respective headings; they are much more frequently met with in their obvious forms in young subjects, and they diminish in frequency and intensity in first attacks occurring after middle-life. The liability, however, to severe heart-involvement, endo-, peri-, or myocardial, increases markedly with each recurrent attack. There cannot be any doubt that often endocarditis is overlooked when the valvulitis is not of such a nature or extent as to cause cardiac murmurs. The presence of a reduplication of the second sound may be the only indication of the temporary damage to the mitral curtains; in severe cases a systolic murmur conveyed towards the left axilla will be present, together with more or less deepening of the constitutional symptoms, especially increase in the pulse-rate, with weakness and some irregularity in the apex-beat. In not a few cases the latent valvulitis only shows itself at a much later period by the results of the injury to the segments causing permanent valvular deformity. Upon the other hand, many cases in which an audible murmur has been present during the attack of fever clear up and show no permanent injury to the valves. In the great majority of cases it is the mitral orifice which suffers; the aortic valves are only affected alone in a small minority, but both mitral and aortic are often involved together.

Malignant endocarditis is rare, except in cases showing old permanent valve-lesions; such cases are prone to develop

serious ulcerative endocarditis in some subsequent severe attack of the rheumatic inflammation. Poynton and Paine have emphasised the fact that an attack of ulcerative or malignant endocarditis may supervene upon an old valvular disease, the supervention being the direct result of an attack of acute rheumatism in which the joint-manifestations may be entirely absent.

Myocarditis is, upon the whole, more likely to appear in its aggravated form in older subjects who have previously suffered from attacks of rheumatic fever; the milder form of the condition may be accepted as the cause of the acute dilatation of the heart before referred to. Undoubtedly myocarditis may have its origin in acute rheumatism without the heart showing signs of endo- or pericardial mischief, as pointed out by Fisher.

Pericarditis, like myo- and endocarditis, may also be latent; it is most frequently observed in very young subjects. When the characteristic to-and-fro friction-sound is audible the diagnosis should be certain. The sudden advent of delirium, with increased frequency, weakness, and irregularity of the pulse, severe precordial pain, anxiety, and increased pyrexia, very usually indicate that the pericardial membrane has become invaded. It may be safely assumed that, as already stated in regard to endocarditis, the presence of pericarditis means that the endocardium and muscular substance are always more or less affected at the same time. Effusion may or may not take place; purulent accumulations are probably the result of a multiple infection. The pain of pericarditis is not to be mistaken for the pain in the epigastric region which so often appears as a symptom of acute rheumatism in children.

Pleurisy and pneumonia are not uncommon; often the pleurisy is an extension of the pericarditis, being confined to the surfaces which overlap the inflamed pericardial sac; but, like the pneumonia, it may supervene when the pericardium is not obviously affected. The lung-complication usually appears in the bronchopneumonic form; but passive congestion from mechanical pressure of pericardial effusion or from cardiac dilatation and acute oedema independent of both these causes are not infrequent. The micrococcus rheumaticus has been demonstrated in the pulmonary lesions and in the pleura.

Nervous disturbances, the most interesting of which is chorea, occur with varying frequency. The connection of chorea with acute rheumatism has been already discussed under the heading of 'Chorea'; the question has been practically settled by the sequence of chorea in animals after inoculation with the specific micrococcus. Multiple neuritis sometimes follows the disease, and may be associated with

considerable local muscular atrophy. Hyperpyrexia is the most serious complication of acute rheumatism, being often fatal; it is probably of cerebral origin, the result of the toxins acting upon the heat-centres. Whilst it is usually accompanied by delirium and ends in coma, both these latter conditions may be present independently of hyperpyrexia; and the same remark applies to the rarer complication of cerebral convulsions, which is sometimes the result of meningeal inflammation.

The throat-complication is frequently present; it usually manifests itself by an erysipelatous redness and swelling, affecting the mucous membrane covering the posterior wall of the pharynx, and causing some œdema of the uvula and tonsils. It is of considerable interest and importance, since the view is widely held that the tonsils afford entrance to the specific micrococcus; but, upon the other hand, the writer has frequently observed that the throat-complication does not show itself till late in the attack, though undoubtedly it sometimes is a well-marked prodromal symptom.

Peritonitis is a rare event, but it has been observed in several cases, and the micrococcus has been found in the inflamed membrane. Albumin is often present in the urine, as in other feverish states, in small amount; but Poynton maintains that a true rheumatic nephritis occurs. Hæmaturia may be sometimes observed in severe attacks of acute rheumatism.

Skin-complications are common independent of sudaminal rashes. The most important of these is erythema multiforme, which occurs in the form of *E. marginatum* and *E. papulatum*, or both combined. With it are often associated distinct manifestations of purpura; the condition is already described and its relationship to rheumatism discussed in the article on 'Purpura.'

Subcutaneous nodules are frequently observed over the sheaths of tendons and under the skin over the fascia covering bony prominences, upon the scalp, and in the neighbourhood of joints. A subcutaneous nodule may occasionally be observed anywhere surrounded by a patch of erythema marginatum. The nodules vary in size from a large pin's head to that of a small bean. The skin is freely movable over them, and the growths may be even made to glide upon the fascia or tendon to a limited extent. They consist of fibrous tissue, with many round or nucleated spindle-shaped cells. Though much more commonly seen in children with established rheumatic valvular lesions and chorea, they are occasionally detected in adults whose hearts remain apparently undamaged. The nodules are often seen to develop in the convalescing stage of the fever, especially when the endocarditis has been severe, and they may remain for several weeks.

Phlebitis is not very uncommon, but the large veins of the legs are not so frequently the seat of thrombosis as are those of the neck and upper extremities, unless in those cases where the inflammation supervenes during the convalescent stage after the patient has been moving about for some time.

Diagnosis.—Usually the clinical symptoms are so well marked that there is seldom a difficulty in recognising the disease. At the beginning of a sudden severe attack there may be, however, a considerable difficulty in eliminating *gout*. The age of the patient, his family-history, and environment afford strong evidence of the gouty nature of a first attack, but much more reliance can be placed upon the localisation of the arthritic mischief. It may be accepted practically as a law that the first attack of acute gout never seizes upon a large joint like the knee, though in recurring attacks every joint in the body may from time to time be the seat of pain, redness, and swelling. The first attack of gout so commonly invades the joint of one great toe that it is only when some other joint chances to be the seat of the mischief that doubt arises. The pain of acute rheumatism while in the mono-arthritic stage, though severe, never approaches to the agony of a typical attack of gout. Clinically, therefore, at the beginning of an arthritis the difficulty only arises in distinguishing rheumatism from an attack of gout in a patient whose many joints had previously been invaded; but the history of the former attacks and the discovery of uratic deposits practically settle the diagnosis. The presence of old tophi cannot, however, be regarded in theory as absolutely clinching the diagnosis of a given doubtful multiple arthritis, since it is conceivable that a gouty patient may become the subject of an attack of acute rheumatism; but such a combination is practically never met with.

Acute rheumatoid arthritis is a rare and terrible disease which in a small percentage of cases cannot for a considerable period of its progress be differentiated from an attack of sub-acute rheumatism. The differential points are discussed under 'Arthritis Deformans.'

Gonorrhœal rheumatism, so called, is recognised by the history of a recent specific infection of the urethra or by the discovery of the discharge. In women the gonorrhœal nature of the arthritis is very liable to be overlooked. Evidences of cardiac mischief of recent origin are commonly absent, but a true gonorrhœal endocarditis undoubtedly sometimes occurs. Iritis, scleritis, and conjunctivitis are liable to accompany the gonorrhœal infection, which is prone to set up a most intractable adhesive type of inflammation. The distribution of the arthritis may assist the diagnosis; though many joints may be involved in the gonorrhœal form, the inflammation does not fly from one joint to another as it does in acute

rheumatism, but tends to remain in the articulation involved. The knee-joint is by far the most frequently invaded, and the synovial effusion may be most extensive. Joints are often involved which are very seldom observed to suffer in acute rheumatism, notably the temporo-maxillary and sacro-iliacs. The adhesive nature of the gonorrhœal inflammation is manifested also in the results of the arthritis; even when suppuration does not occur ankylosis very frequently follows.

Septic arthritis is the name given to a condition closely allied to gonorrhœal rheumatism. It is the result of infection by pyogenic microbes or by the organisms which cause the exanthematous fevers, erysipelas, influenza, &c. The arthritis is multiple and often suppurative; the cardiac, cutaneous, and other manifestations of acute rheumatism are absent, the clinical picture being that of the underlying septicæmic disorder. *Pyæmic* joint-trouble is always purulent, is ushered in with a succession of severe rigors, and is usually accompanied by metastatic abscesses elsewhere, forming a group of symptoms quite different from the transient multiple arthritis seen in acute rheumatism. Midway between the septicæmic and pyæmic types of multiple arthritis is seen the form of joint-mischief which sometimes supervenes upon severe ulceration of the colon in dysentery. Like a similar septic form of osteo-arthritis not rarely met with after parturition, the affection may closely simulate for a brief period the opening chapter in a severe attack of acute rheumatism, but the underlying septic state should easily serve to exclude the latter affection.

Acute necrosis is only liable to be mistaken for rheumatic fever when the osteo-myelitis is in its early stage or in the rare infective form in which it is multiple. The deep tension, causing great tenderness and swelling amounting sometimes to œdema of the entire limb whose long bone is affected, is quite distinctive. In all septic forms of arthritis, except during the perspiration which immediately follows the rigors, the skin is burning, hot, and dry, in marked contrast to the continual moist sweating of the rheumatic condition.

'*Growing pains*' in children are often, if not always, a sign of rheumatism, and should direct immediate attention to the state of the heart.

In connection with the diagnosis of acute rheumatism the discovery of the subcutaneous nodules may practically be regarded as settling the nature of the morbid process. Hardly less conclusive is the remarkable relief almost invariably obtained by full doses of salicylates; this method of making a therapeutic diagnosis approaches to the value of a certainty in the differentiation of gonorrhœal synovitis, which invariably fails to respond to salicylate-treatment. Terc has found that the latter condition may be distinguished

from acute rheumatism by causing a bee to sting the patient : in rheumatism the bee-sting always produces a non-inflammatory vesicle, and it is well recognised that bee-keepers seem to be more or less immune to rheumatism.

Prognosis.—The direct mortality of the disease is very slight considering the serious conditions which are often present ; only a trifling percentage of patients succumb during the attack—probably not more than 2 per cent. of the whole. The cause of death is usually the carditis. The most fatal complication is hyperpyrexia, but by prompt treatment even this formidable condition may sometimes be successfully dealt with. Most authorities agree that the presence of numerous subcutaneous nodules, especially in children, is an omen of grave significance, being often indicative of progressive fatal destruction of the valves. The prognosis of acute rheumatism is always more serious in the case of children than in adults. The forecast as regards the question whether obvious involvement of the heart may be expected in a given case cannot be attempted ; more than half the cases develop signs of carditis. A more important and practical element in prognosis is the question of how far the subject of acute rheumatism suffering from carditis is likely to eventually become the victim of permanent valvular disease. Upon this point it may be said that, whilst drug-treatment in the fever-stage does not appear to markedly affect the final result, the liability to permanent valve-deformity increases in proportion to the shortness of the rest-cure.

Treatment.—Immediate rest should be enjoined in all cases as soon as the diagnosis appears even probable. Early resort to bed and prolonged rest after the disappearance of the last arthritic manifestation is of vital importance, and it is well to let the patient recognise this inevitable restriction at the beginning of the treatment. He should not be placed on a feather-bed, but upon a smooth soft hair-mattress, and sheets (either linen or cotton) must be discarded, a soft blanket being placed beneath him and another thin one above him. His bed-dress should be loose and of thin wool ; the bed-clothes also should be light, but the objectionable eider-down coverlet must be condemned, as it tends to increase the sweating by retarding evaporation.

The best diet is a liquid one ; milk in most cases is all that is necessary, but vegetable and animal soups and light farinaceous foods may be permitted from time to time. Alcoholic stimulants, as a rule, are unnecessary and often injurious, except as a means of combating heart-failure. Iced water may be permitted in moderation when thirst is severe, but a better plan is to dilute the milk with an alkaline effervescent water and keep the mixture cool by constant immersion in an ice-bath.

Local treatment may be initiated from the start, the affected joints being best treated by a thick layer of absorbent wool held in place by a thin open-webbed bandage ; splints and other motion-restraining appliances are usually annoying and unnecessary. When the pain is very severe a layer of lint saturated with chloroform-liniment or methyl-salicylate may be applied under the cotton-wool ; but impervious covering should be discarded owing to the abundant perspiration. Blisters over the affected joint or, better still, a ribbon of cantharides-plaster encircling the limb above and below the joint, often proves efficacious in the relief of pain. Osler prefers a light application of the thermo-cautery. The writer has no practical experience of cold applications to the affected joints as praised by many Continental physicians.

The old alkaline treatment, lemon-juice, quinine, iron, and other forms of drugging, have given way to the administration of salicylate of soda ; mercurialisation, purgation, and bleeding are now entirely abandoned. The salicylate treatment in the great majority of cases gives prompt relief to all the arthritic manifestations of the disease. Under its use relapses are said to be common, but this is probably an error. The patient after a few days' relief from all joint-pain can sometimes be restrained with difficulty from sitting up or getting out of bed, with the result that the joints become again painful, the symptoms having been masked by the analgesic action of the drug. In this way the salicylate-treatment may prove harmful by deceiving the physician, who does not realise that it is in no true sense a specific for the disease. When employed simply as an empiric agent for the relief of pain and fever, in combination with prolonged rest, its benefits cannot be exaggerated, and it probably, when this latter precaution is rigidly carried out, shortens the real duration of the attack and possibly lessens somewhat the liability to serious cardiac involvement.

Relapses frequently occur in acute rheumatism, and these are but slightly prevented, if at all, by drug-treatment ; they are probably due, as pointed out by Carey Coombs, to the micrococcus persisting in a quiescent state in the affected tissues. Movement of the invaded joint or increased cardiac action may arouse the virulence of the organisms, so that from first to last the rest-element in the management of an attack of acute rheumatism in all its stages should receive the fullest recognition. Salicylic acid, salicin, oil of winter-green, and the more recent derivative aspirin or salicylo-acetic acid act in the same way as the soda-salt. Aspirin is preferred by Mackey and others, since it passes unchanged through the stomach to be absorbed slowly by the intestine, producing a more continuous effect than the more soluble salicylates. For routine treatment 20 grs. of the purified

salicylate of soda administered every two to three hours answers all purposes admirably, the interval between the doses being promptly lengthened as the fever and pain subside. The newer analgesics—antipyrin, phenacetin, &c.—reduce the fever promptly, but they cause increased drenching perspirations, their effects are evanescent, and they depress the heart seriously when continued for any length of time.

The treatment of the carditis endo-, peri-, and myo-, is to be carried out upon the same principles, details of which are found under their respective headings. As a rule the salicylate administration may be continued in the presence of the cardiac symptoms, but the dose must be lessened and its action closely watched, the drug being suspended upon the appearance of marked heart-weakness or threatening failure. Under these latter circumstances, with the joint-pains continuing, the writer has often with advantage administered pure salicin, alone or in combination with quinine.

Of all the complications of acute rheumatism, hyperpyrexia demands the promptest treatment. Antipyretics and salicylates are not to be depended upon, the only reliable agent being the free use of cold water. The patient should be quickly lifted into a cold bath, or placed upon a waterproof mattress, and enveloped with a large wet sheet, over which cold water is poured till the body-temperature falls to slightly above the normal.

With the recent recognition of the infective nature of the acute rheumatic process more scientific methods are being eagerly sought after. Schäfer and Schmidt have tried the antistreptococcic serum of Menzer; although the results are not brilliant, they nevertheless warrant a hope that by improved methods a serum may ultimately be produced by injection of animals with gradually increasing doses of cultures of the specific micrococcus or of its toxins. Menzer's serum is prepared by injections of cultures of streptococci obtained from so-called rheumatic sore-throat; it certainly appears to be harmless. Its introducer maintains that its efficacy is a proof that acute rheumatism is a non-specific disease, but its 'efficacy' yet remains to be demonstrated.

ACUTE RHEUMATISM IN CHILDREN

The clinical manifestations of the disease in subjects of tender years differ considerably in degree from those which constitute the typical picture of acute rheumatism in adults. The main differences have been incidentally noticed in the foregoing description, but for convenience they may be briefly recapitulated. The onset is as a rule seldom abrupt in young children, there being usually a history of more or less vague joint-pains for some days or even weeks before the child takes to bed. These, in the majority of cases, are

regarded as 'growing pains.' It is a good rule to regard all such cases as examples of rheumatism, and to investigate the family-history carefully, as heredity is a more important etiological factor in the rheumatism of children than in that of adult life; not infrequently an hereditary history will be forthcoming on the part of both parents.

The pyrexia is usually much lower than in the case of adults, with the exception that where pericarditis supervenes it is very high; but sometimes even severe endocardial mischief may exist with a slight degree of fever. The joint-manifestations as a rule are less marked and more transient; in all other respects as regards pain, heat, redness, swelling, and distribution, the conditions are identical. Pain and stiffness of the neck-muscles and epigastric pain are much more constantly present than in adults. Sweating is decidedly less in children, the skin often remaining dry, unless diaphoretic treatment has been pushed; and the sour odour of the perspiration is rarely detected. Sore-throat with considerable swelling and pain in the tonsils is very commonly present in the early stages, and may remain throughout the attack. Pleurisy is as frequent a complication as pneumonia is a rare one. As in adults, the pleuritis is commonly of the plastic type, but copious effusion is not infrequent; the inflammation in the majority of cases is an extension from the pericardium. Chorea is very often associated with the rheumatism of childhood; perhaps half the cases may be found either to afford a previous history of this complication, or to be associated with the attack, or to develop at a subsequent period after convalescence. Carditis is much more prone to be a severe and damaging element in the rheumatism of childhood, and in this mainly lies the difference between the manifestations of the disease in adults and young children. Probably four-fifths of all patients under six or seven years of age suffer from obvious endocarditis or pericarditis, the latter condition being less frequent than the former but very much more frequent than in the case of adults. Myocarditis is also evident in all fatal cases, and probably it is present in some degree in every case. Its clinical manifestations are, however, not so perceptible, but the acute dilatation of the left ventricle which, according to Lees, is almost constantly to be detected in attacks of acute rheumatism in children, appears to be certainly the result of a rapid deterioration of the cardiac muscle, either from the influence of the toxins directly or from the intense and rapid myositis which sometimes follows.

The frequency and severity of the carditis in young children, even when the joint-manifestations are comparatively transient or evanescent, support the generally accepted view that a considerable proportion of the cases of valvular

disease found in young subjects, in whom no history of rheumatism is obtainable, are due to an attack of the disease in which the arthritic manifestations were absent or so trivial as to be overlooked.

Subcutaneous nodules are very frequently present. Still found them in almost 50 per cent. of his cases; they may be detected in any of the situations already mentioned. Coutts emphasises the fact that the back of the elbow-joint is by far their most frequent site. Their presence may be safely accepted as evidence of serious implication of the cardiac valves, and Barlow and Warner maintain that they are analogous to or identical with the nodules found on the margins of the mitral curtain. As already stated, their presence, when of large size and in considerable numbers, is a very grave omen in prognosis, and should the patient recover they may be regarded as evidence of the extreme probability that permanent valvular deformity will follow.

Skin-rashes of the erythematous multiform type, associated with purpuric manifestations are more common than in the adult, and when profuse are almost invariably accompanied by the presence of nodules. A fugitive diffuse scarlatinal roseola is sometimes observed in the early stage of the disease, but the subsequent progress of the case leaves little difficulty in excluding scarlet fever. As will be mentioned under 'Scarlatina,' a rheumatic form of arthritis is often present in that disease. A similar eruption has been sometimes observed in poliomyelitis anterior acuta, in which a mono-arthritis has been also present, but the paralysis and rapid local wasting speedily assist in the recognition of the spinal affection.

RHEUMATISM, CHRONIC

When an attack of acute rheumatism appears in mild form, with a sub-febrile temperature and slow onset, the affection is often catalogued as Sub-acute Rheumatism. Such an attack is doubtless liable to run a much slower course, but in the manifest identity of the disease with rheumatic fever there is usually little room left for doubt, as the arthritic involvement resembles that seen in the acute affection, and the frequency with which cardiac and other manifestations occur establishes the unity of the disease. Moreover, sub-acute attacks of articular rheumatism, lasting sometimes for months, occasionally are witnessed in subjects who have previously passed through a typical attack of the acute disease, and sometimes such attacks alternate with those of rheumatic fever.

The aspect of the clinical problem changes, however, when the consideration of so-called *chronic rheumatism* is approached.

The affections formerly recognised under this title have been steadily relegated one by one into such categories as rheumatoid arthritis, gonorrhœal synovitis, morbus coxæ senilis, pulmonary osteo-arthropathy, septic, tubercular, and syphilitic arthritis, coxa vara, flat foot, &c., until it would appear to the superficial observer that there is left no such entity as chronic rheumatism; neither can there be in the true sense. If acute rheumatism be accepted as an infective disease, caused by the invasion of a specific microbe, then, as forcibly put by Stockman, it cannot be considered as becoming chronic any more than diphtheria or typhoid fever becomes chronic. These diseases may leave sequelæ behind them, of long or indefinite duration, after the germs which caused the diseases have perished or ceased to manufacture their toxins in the body; hence the painful arthritic, fibrotic, and valve-changes left after an attack of acute rheumatism must be regarded as sequelæ and not as a chronic stage of the disease. The difficulty in clearly recognising this important distinction has probably arisen from the fact that acute rheumatism is a disease prone to frequent recurrences. A mild attack or relapse, supervening upon joints which have been left painful and stiffened, or upon valves which have suffered damage from a recent attack, is very liable to be regarded sometimes as a chronic type of the infective disease, in cases where a true convalescence had apparently never been established.

It therefore makes for progress if all cases definitely arising from or following an attack of acute or sub-acute rheumatism be excluded entirely from the title of 'chronic rheumatism,' even should subcutaneous nodules of very long duration be found present. When this is realised, there is certainly left an affection of somewhat common occurrence, which is entitled, from the pathological and clinical standpoint, to the designation 'chronic rheumatism.'

Pathology.—This has been ably worked out by Stockman, who finds that the essential pathological changes are confined to the white fibrous tissue entering into the structure of the articulations, of the tendons and their sheaths, the aponeuroses, periosteum, septa and sheaths of muscles, nerves, &c. The process causing the lesion consists of an inflammation leading to hyperplasia of the connective tissue, which occurs in patches, the original irritative factor being probably a toxæmia entirely different from that caused by the toxins of the micrococcus rheumaticus. This latter attacks the articular structures proper—cartilage, synovial membrane, and occasionally bone—while the poison of chronic rheumatism exercises its selective influence over the white fibrous tissue of the capsule, ligaments, tendons, muscles, and nerves. The process in its acute phase is of the same nature

as the fibrositis described by Gowers, and referred to under 'Lumbago.'

The hyperplastic indurations or patches of Stockman are frequently circumscribed; like the subcutaneous nodules of acute rheumatism, they may reach the size of a small almond or half a walnut, but, as he points out, a whole aponeurosis may become indurated, or a cord or strand of thickened fibrous material may run through its entire length. In some cases the induration may involve a considerable portion of a nerve- or muscle-sheath; no leucocytes are found in the thickened tissue, which, however, is well supplied by blood-vessels. The indurations may entirely resolve, but they are liable to swell up and become very painful at times upon exposure to cold, muscular action, or injury of any kind.

This is apparently the pathological condition so often witnessed after fractures, sprains, and severe contusions, and whenever a joint has long ceased to be subjected to the normal stimulation of healthy movements. The indurations, when superficial, are somewhat tender to pressure and are most frequently to be detected in the lumbar aponeurosis, calves, fascia lata of the thigh-muscles and glutei and of the shoulder-girdle muscles, and in the plantar surface of the feet.

Etiology.—Stockman differentiates between the original causes of the fibrous indurations and the etiological factors which excite severe exacerbations of pain, and the other manifestations of chronic rheumatism. The primary cause of the fibrositis may be injury, as in fractures and sprains, or it may sometimes show itself as a sequela of acute rheumatism, influenza, or sore-throat; often no etiological factor can be discovered except severe or long-continued exposure to cold, damp, or variations of temperature, often in association with severe muscular strain. After the formation of the induration has once become established, exposure to cold and damp usually causes the newly formed tissue to swell, and the swelling produces by its increased tension severe exacerbations of aching pain through nerve-pressure. Variations of season and sudden meteorological changes appear to act in a similar manner, even when the patient is confined to bed for other reasons. The condition is rare in children; its first manifestations usually show themselves about early middle-life. Owing to its chronic course its incidence is usually associated with the idea of senility, in which respect it somewhat resembles gout, with which, however, it has no necessary pathological connection.

Symptoms.—These depend upon the region whose white fibrous tissue has been involved. When the fascia of the lumbar area or of the neck, intercostal, or shoulder muscles suffers, the symptoms may in the first instance appear in an acute form, constituting lumbago, stiff-neck, pleurodynia,

scapulodynia, or other form of so-called 'Muscular Rheumatism' or Myalgia or Myositis, but the natural tendency of the fibrositis shows itself by its recurrence and in its inclination to become chronic. In all the above regional varieties the affection may commence in a sub-acute or typically chronic insidious manner. The most obvious symptom is pain; this may be a dull continuous aching, subject to exacerbations, and always increased by active muscular movement, which reveals painful stiffness, sometimes almost amounting to fixation, as in lumbago. In mild cases there is no spontaneous pain, and the pain brought on by movement gradually fades as motion is continued, though the muscles are easily exhausted; but after the cessation of movement the aching frequently returns with increased severity for a time. Sudden movement always brings on an acute exacerbation of intense pain, whether the affection be confined to the neighbourhood of a joint or to the lumbar or other fasciæ. There are as a rule no constitutional disturbances; even in apparently acute severe cases the temperature is normal and the circulation uninfluenced. The digestion is not infrequently disturbed, but often this may be found to have preceded the attack, and probably was its exciting cause. Gentle pressure usually gives some relief, so that patients may often be observed stroking or rubbing the affected muscles or joints during an exacerbation. The chief diagnostic sign should always be sought for in the presence of the indurated patches, which, however, are often but slightly tender under even a considerable degree of pressure, unless during the acuter exacerbations. A true neuralgia is said to be sometimes associated with the more chronic forms of the disease, but neuritis is a more common sequence from the fibrositis of the nerve-sheaths. The presence of either condition tends to obscure the diagnosis. The skin should be smeared over with vaseline, olive oil, or liquid paraffin, the muscles placed in a passive state, and the entire painful region gently but very thoroughly explored by the finger-tips for nodules, patches, indurated cords, or more diffuse infiltrations.

When the pain is localised in the vicinity of a joint, careful examination will usually satisfy the physician that the seat of the mischief is outside the articulation. The absence of the marked anatomical changes in the cartilages and bones at once eliminates *rheumatoid arthritis*, and the absence of urate of soda concretions disposes of *gout*.

Prognosis.—The outlook in chronic cases, with considerable fibroid induration, is very gloomy as regards recovery, though there is no danger to life. Even when by active treatment the local condition is removed, there is always the great probability of a return of the disease on some trivial exposure, sprain, or failure in health. The prognosis in the

acuter forms of muscular rheumatism is decidedly hopeful in first attacks, but each recurrence brings the affection nearer to the typical chronic form.

Treatment.—Drugs are of little value save for the alleviation of the pain in the acute exacerbations of the disease, when absolute rest is also essential. Salicylates and aspirin in full doses relieve, like other analgesics, but they are in no sense curative. For routine administration, full doses of iodide of sodium give the best results in combination with guaiacum.

Massage is by far the most useful agent when persistently and conscientiously carried out, the indurations being subjected daily to a gradually increasing amount of forcible manipulation. A warning must, however, be sounded in reference to the abuse or unskilful application of this valuable agent; the writer has seen severe intractable neuritis suddenly set up in the affected parts by a vigorous massage commenced without due gentleness. Passive movement in the variety of the disease in which the fibrositis is confined to the neighbourhood of articulations often acts like magic; the value of this agent is demonstrated in the chronic rheumatism which follows a recent fracture. Electricity in the form of the induced current may be advantageously applied in conjunction with massage, passive movement, and gymnastic exercises. All these agents can be utilised, in combination with hydropathic treatment, or in conjunction with a course of brine-baths at Droitwich or Nantwich. An alkaline spa in some cases, and in others a course of natural sulphur-water, are valuable adjuncts. Blistering the affected parts aids in the removal of indurations, but interferes with the use of massage and passive movements. As a rule, the application of warmth to the affected muscles and joints by flannel bandages, cotton wool, &c., is carried out by the patient to an injurious extent. By heaping on excessive clothing the disease seems often to be fostered. Residence in a dry, moderately warm, but not hot climate, at a distance from the sea, is always beneficial. Stockman advises surgical methods for the extirpation of nodular masses which resist massage.

RHINITIS. (See under CORYZA, p. 245.)

RICKETS, or RACHITIS

This is a chronic disease affecting children usually between the ages of six months and two years, characterised by marked alteration in the general nutrition, mainly exhibiting itself in enlargement of the epiphyses and softening of the

bones, which often eventuate in characteristic permanent deformities. It is sometimes also called the 'English Disease.'

Etiology and Pathology.—The disease is widespread, but chiefly abounds in the densely populated cities of cold and temperate climates, being rare in warm regions and in rural districts. Few diseased conditions have given rise to so much speculation as regards etiological considerations. Many factors have been demonstrated to be constantly present, but possibly the real causal agent is still awaiting demonstration, the various factors now recognised as causes being but predisposing agents. Impure air, improper food, poverty, absence of sunlight, and other hygienic deficiencies may produce their ill effects upon the infant's mother before its birth or during lactation. The latter process, if carried on during pregnancy, may predispose the fœtus to the disease, whilst it may act at the same time as an exciting cause in the child receiving nourishment from the breast.

Male infants suffer as often as female; very few cases appear before the sixth month or after the twenty-fourth. So-called 'late rickets' will be referred to further on. Heredity is held to play no part in the causation of the disease, but recently Seigert strongly contends that the opposite is the fact. There cannot, however, be a doubt that all forms of hereditary or congenital weakness tend towards predisposing the weaklings to rickets. The unhealthy environment already mentioned as liable to vitiate the health of the mother and her milk may also directly operate upon the infant who is artificially fed from its birth. It is in bottle-fed children that the disease nearly always is observed, and especially in those brought up upon artificially prepared starchy foods and condensed milk, but the disease is common amongst infants improperly fed upon the fresh milk of apparently healthy cows.

It would appear that the nature of the food is the strongest acting etiological factor, but many authorities only regard the nature of the food from the standpoint of its dyspepsia-producing powers, believing that rickets is but the result of a chronic dyspepsia. Ashby considers that the dyspepsia leads to the production of bacterial toxins in the intestine, which, being absorbed, cause the disease. Others maintain that the dyspepsia leads to greatly increased production of lactic and diminution of the hydrochloric acid. The paucity of hydrochloric and the excess of lactic acid are supposed to interfere with the assimilation of the lime-salts. Cheadle's view that rickets is produced by the absence of fat in the food is widely held, and receives confirmation by Bland Sutton's success in preventing rickets in the offspring of lions and bears, reared in captivity, by adding animal fat to their diet, in combination with minced bone. The disease is, however,

often observed in infants when the amount of fat in the food has been such as maintains other infants in a perfectly healthy condition. To meet this latter difficulty in the rickets-puzzle, Still's theory is probably sound, and may be accepted at present as the best working hypothesis of the etiology of the disease. According to this observer, the chief dietetic factor in the production of rickets is the 'deficiency of fat-assimilation, whether from the paucity of fat in the food, or from digestive disturbance, interfering with its absorption.' When there is excess of carbohydrates in the food, this interferes with the absorption of the fat, so that a diet including enough fat for the requirements of the organism may produce rickets when excess of carbohydrates are added to it. Freeman explains the rarity of rickets in warm climates as due to the better oxygenation of the blood, since the infants spend most of their time in the open air; but the explanation may possibly lie in the diminished necessity for fats under such environment, or in the absence of the dyspeptic troubles which affect children brought up in the vitiated air of cold, damp, overcrowded city dwellings. The view that rickets is caused by syphilis is no longer tenable.

Morbid Anatomy.—The most obvious changes are found in the bones. The long bones of the limbs especially, and the ribs, are involved, but all the bones show marked changes. The epiphyses are swollen, the periosteum is thickened, there is great deficiency of lime-salts, as proved by chemical analysis, and there is excess of the red, grumous, medullary material. The entire bone is soft, and may be bent easily by approximating its ends; sometimes it can be readily broken by pinching it between the fingers. At a further stage of the disease the imperfectly ossified bones will be found curved or bent in various ways, whilst later still they may be seen ossified in their abnormal curves and displaced fractures into dense ivory-like tissue.

Upon examining a long bone, removed from an infant succumbing to any of the complications of severe rachitis, the swelling or bulging at the epiphysial extremities is an obvious feature. It has been apparent during life, especially over the lower ends of the radius and ulna, and the lower end of the tibia, and the beady swellings observed at the junctions of the ribs with their cartilages, are of the same nature. This swelling is seen, upon longitudinal section, to be situated between the shaft and the epiphysis in the columnar layer of the cartilage, where the proliferating zone is found thickened and with its matrix undergoing softening. Calcification is found to be proceeding in a very active but irregular manner in some of the cartilage-cells, whilst it is delayed in others and in the trabeculæ. The epiphysial layer lying beneath the proliferating zone is very much broadened and

irregular, instead of presenting the normal appearance of a narrow clearly defined line or band. The process of true ossification appears to stop short, after exhibiting premature proliferation of the cartilage-cells, so that a soft, imperfectly formed, cancellous tissue results. There is increased vascularity or hyperæmia, so that new bone is not laid down as fast as already formed bone is rapidly removed by absorption. The thickened periosteum is easily peeled off, and immediately under it a layer of bone is found in the same vascular, spongy, soft, cancellous condition, exhibiting premature or precocious proliferation and retarded deposition of lime-salts. The flat bones of the cranial vault are soft and spongy also; they cut easily with a knife, and bend under the pressure of the fingers. The irregular manner in which the calcification has proceeded is here again evident; the frontal and parietal eminences, where ossification had first started under the early normal conditions, show considerable flat hypertrophic thickening, whilst atrophic thinning is apparent in other parts of the same bone, so that at these spots it may seem transparent when held up to the light. During life these thinned-out areas may be made to emit a peculiar crackling sound when indented by the pressure of the finger, like that caused in a thin tin vessel upon rapid removal of the finger-pressure after pressing-in a limited area upon the sides or bottom.

The nature of the changes in the bone is obviously secondary to the blood-condition, and some authorities regard them as of an inflammatory type; certainly the hyperæmia is a marked feature in all the region about the proliferating zone, in that underlying the periosteum, in the periosteum itself, and in the red marrow.

The muscles share obviously in the general impaired nutrition of the body; they are soft and wasted. The flabby state of the atrophied skin, which hangs at places in pendulous folds, is equally characteristic of the abnormal state of the general nutrition. The liver and spleen are enlarged, and often the mesenteric glands. Complications commonly present in all fatal cases cause changes of the bronchopneumonic type in the lungs and catarrhal thickening of the gastro-intestinal membrane. The blood is deficient in hæmoglobin and red cells in many cases, especially when there is obvious splenic enlargement; there may be marked leucocytosis, and nucleated red corpuscles are sometimes present in considerable numbers.

Symptoms.—Before any marked bone-changes can be detected there are generally significant symptoms which are liable to be overlooked. Digestive disturbances are nearly always present, as seen in flatulent distension of the abdomen; the child is restless, always sleeps badly, kicking off the bed-

clothes ; the skin of the body is moist and clammy ; profuse beady sweating is almost constantly observed about the head and neck, which saturates the pillow-covering, as the back of the scalp is continually pressed or rubbed against it both in sleeping and when awake. Sudamina are common. There is usually a febrile temperature, but, unless complications occur, the thermometer seldom registers beyond 101° .

A feature of great diagnostic importance during the early stage is pain in the limbs and bones. This symptom has been the subject of considerable difference of opinion ; some authorities (Colman and Czerny) deny its existence in simple uncomplicated rickets, and maintain that pain and tenderness in the bones are always a sign that scurvy is present. Jenner regarded it as a very early and important sign of rickets ; and Zanetti, having closely investigated the symptom, holds that all rachitic children suffer pain in the bones, which sometimes is very severe : the child lying quiet with its flaccid limbs at rest screams when the bones are pressed upon or even moved. As a rule, the signs of failing nutrition soon develop ; often anæmia appears, and some signs of wasting, associated with softness of the muscles and obvious signs of loss of power in the limbs ; but it is difficult to determine how far the latter is due to the inhibitory influence of the pain.

These early symptoms may be associated with convulsions, which are liable to be referred to the delayed dentition. Convulsions occurring at a later stage may be due to the changes in the skull-bones, but it cannot be doubted that the rachitic condition, independent of complications, is, as maintained by Thomson, a powerful predisposing cause of convulsions.

Another early diagnostic sign is pointed out by Francine, who found it present in 70 per cent. of cases, and styled it 'diastasis of the rectic muscles.' It is caused by the bulging forwards of the distended abdominal contents, which cause separation of the recti abdominis muscles, whose tone has become lowered.

All the above symptoms may be marked before the advent of the sixth month, but as a rule they appear after this period, and are followed within some weeks or a few months by the signs of the bone-mischief.

The first change in the skeleton is usually seen in the enlargement of the epiphysial ends of the long bones, which may be detected readily on inspection and palpation in thin infants by the beadings, or so-called 'rosary,' which occur upon the ribs at their junctions with the costal cartilages, extending commonly from the fourth to the ninth. These may in rare cases be found as early as the fourth month, and may continue to increase till the end of the normal dentition-period. The

ribs and cartilages, owing to their softened condition, yield in inspiration, so that marked deformity is produced, which usually remains more or less obvious through life. The cartilages and sternum are thrust forwards, causing a type of pigeon-breast, but the protuberance is rounded and does not show the sharp keel which characterises the true pigeon-chest; the latter, as pointed out by Ashby, is the product of changes in the cartilages which occur from other causes at a later period of childhood. A broad, shallow, vertical or oblique groove is formed on each side of the sternum by the falling-in of the ribs during inspiration. Sometimes a horizontal sulcus, known as Harrison's groove, is permanently formed, which passes transversely outwards from the lower extremity of the sternum; it corresponds to the attachment of the diaphragm. With it the lower margin of the thorax is everted, and as the diaphragm is brought into action the chest-wall is pulled inwards, causing deepening of the sulcus in inspiration.

The long bones in the upper and lower extremities usually show the enlargement of the epiphyses at the wrists and ankles. The lower ends of the ulna and radius are distinctly enlarged, as is also the junction of the epiphysis of the tibia with its shaft.

Much interest attaches itself to the bones of the fingers, whose condition has only attracted minute attention within the past few years. Here, instead of the epiphysial swelling, the diaphysis is increased in girth so that each phalangeal bone, instead of presenting a drum-stick appearance, assumes a spindle-shape owing to the proliferation of the layer lying below the periosteum covering the phalanx.

The changes in the cranial bones are as a rule evident before those in the bones of the limbs; the parietals and upper portion of the occipital may be found to be thinned out at places so that they readily yield to the pressure of the finger, causing the condition recognised as *cranio-tabes*, which is, however, also observed in syphilis, whether associated with rickets or not. The fontanelles remain long open and very large, but the most characteristic change in the skull is the thickening, or hyperplasia, which is observed over the prominences of the frontals and parietals; the bosses on these, however, do not consist of healthy firm bone, but of a very spongy osseous tissue. The enlargement of the prominences gives a *square* appearance to the skull, which looks considerably bigger than that of a normal child, but the appearance is deceptive owing to the fact that the facial bones are stunted; the skull is, however, really somewhat increased in circumference.

Another type of deformity is less frequently met with; this is known as the *natiform* skull, the enlargement being in the antero-posterior diameter, though the vertex is still

flattened and shows two broad grooves, one over the longitudinal, which crosses the other situated over the transverse suture. It only occurs in severe forms of the disease. Both these conditions may be found associated with circumscribed small areas of atrophic thinning, in which nothing but membrane seems to exist; and with them may be sometimes seen obvious flattening of the occipital bone, over which the scalp is often devoid of hair owing to the constant boring of the head upon the pillow. The skin is thin and parchment-like, showing full veins, the whole physiognomy forming with the small face a highly characteristic picture.

With the above signs are usually associated some anæmia and general wasting; but the opposite condition is not rare, the child long remaining fat and even rosy. Almost invariably the dentition is delayed and irregular: the first tooth often does not appear till the end of the first year; the eruption generally does not proceed in the usual regular order, often the teeth are irregular in shape, and they soon decay.

If the disease has appeared early the characteristic deformities of the bones are delayed, save in the cranium and thorax. These deformities are caused by the weight of the soft parts as soon as the child commences to crawl or walk, hence they do not become marked till well on in the second year. At the termination of the first year the child is usually unable even to sit up upon its mother's knee owing to the weakness of the muscles, and it may be another six or nine months before it shows ability even to crawl or walk; by this time the spinal column may be very markedly curved in the vertical direction. Probably also the bending of the bones is effected to some extent by the action of the muscles. With attempts at crawling along the floor the weight put upon the arms causes bending or bowing of the fore-arm and upper-arm bones, which bow outwards. The tibiæ and fibulæ curve inwards and the femora bow outwards and forwards, and the neck of the latter bone is liable to bend, causing the coxa vara deformity. The upper extremities may be markedly deformed in children who make no attempt at locomotion, but who simply sit on the floor supporting the trunk by the hands resting on the carpet. The pelvic bones suffer later on and may cause serious permanent deformity, which tells at the obstetric period. Knock-knee is liable to develop as soon as walking becomes possible, which may be delayed till after the third year.

The late stage at which walking is attempted is apt to cause an exaggerated conception of the general 'backwardness' of the child, but there is good reason to believe that always some retardation in the mental development takes place, though the connection between permanent feeble-mindedness and rickets cannot be regarded as established.

As already stated, however, convulsions are liable to occur in rachitic children; restlessness, bad temper, and insomnia are common in some, whilst others are lethargic and dull, and the majority perhaps show considerable difficulty in learning to speak. Tetany is not uncommon, and laryngismus stridulus is often observed.

As regards the locomotor system, it must, however, be remembered that the skeletal changes never exist alone: the muscles and ligaments always suffer; indeed, every tissue in the body seems to be impaired in its nutrition and retarded in its development. This is seen in the stunted or dwarfed dimensions of rachitic subjects, whose stature sometimes, independent of the reduction caused by bendings, is considerably lessened.

The deformities are sometimes greatly intensified by fractures; the softened bones are often very fragile, so that 'green-stick' fractures occur, which are liable to unite at various angles, the broken bone being held in its abnormal position by the thickened periosteum.

In addition to the above symptoms, the subject of rickets is very liable to suffer from bronchial catarrh and catarrhal pneumonia, which may prove fatal; probably more or less gastro-intestinal catarrh is always present, and should be regarded as a symptom of the disease and not as a complication. It is evidenced by the protuberant distended abdomen, which is almost invariably present and which contrasts so markedly with the deformed chest and stunted body.

Scurvy is a serious complication of common occurrence, and, as already stated, it is believed by many authorities to be always present when there is marked tenderness of the bones. The enlargement of the spleen, and anæmia, have been referred to under the Pathological Anatomy of the affection.

The duration and course of the disease vary considerably. When the condition appears in mild form it may entirely escape observation. Even moderately severe cases may so resolve that little or no deformity of the skeleton remains, the bones becoming finally normal in every respect save that there may appear to be some exaggeration in their natural curves. Softening of bones occurring in children who have been carelessly permitted to walk and crawl about produce marked and obvious deformities, which cannot be expected to resolve themselves without surgical aid. Occasionally the square head, deformed thorax, and bowed limbs may be seen in late life, as evidently marked in the firmly set condition of the bone, as during the second or third year of the child's life; such cases suffer usually also from knock knee, flat foot, and other results of stretching of the ligaments. The state of the urine is normal in the majority of cases; rarely is any excess of lime-salts to be detected at any stage of the disease.

Fœtal Rickets is the name given to a condition supposed to be closely allied to the ordinary form of the disease, in which fractures occur during or soon after delivery, or the infant may exhibit at birth bones already softened and even bent. Such cases, though there may be enlargement of the epiphyses and beading of ribs, are held by many authorities to be of different pathological causation from true rickets, some maintaining that the skeletal changes are simply due to deficiency of lime-salts—a condition which does not occur in rickets; others regard them as allied to cretinism or achondroplasia.

Late Rickets is also a by no means clearly definable condition. Undoubtedly, but extremely rarely, true rachitis does supervene in late childhood or early adult-life with all the typical features which the ordinary disease shows at the end of the first or during the second year. Some of these rare cases are supposed to be the result of a relapse in a subject whose primary rachitis was so slight as not to have been observed. The relapse (if it can be called such) may pursue a very chronic course, lasting for several years and terminating in severe permanent deformities. Some cases reported as late rickets appear to be examples of osteomalacia.

Diagnosis.—In a child at the usual rachitic period—*i.e.* between the ages of six months and two years—the early symptoms (restlessness, feverishness, sweating, kicking off of the bedclothes, and diffused soreness of the body), as pointed out by Jenner, should sound the alarm of the advent of rickets and cause daily examination of the cranial and long bones. Once the changes in these have developed there can be no longer a doubt about the diagnosis. It must, however, be kept in mind that in mild cases, only some of the bones may show obvious alterations; thus the cranials are sometimes alone affected, at other times the beading of the ribs and chest-deformity only may be obvious, and in not a few cases little can be made out save the epiphysial swelling above the wrists. In all cases of marked delay in the dentition, especially if associated with laryngismus stridulus, rickets should be suspected. The advanced disease is recognised at a glance—the small face, large square head, stunted chest and limbs, the pot-belly and separated recti muscles, spindle-shaped phalanges, and enlargements at the ends of the curved bones form one of the most characteristic pictures in clinical medicine.

Infantile paralysis is simulated at the stage when the muscular weakness shows itself most, but careful examination will reveal in rickets the uniform distribution of the weakness and tenderness, the absence of abrupt marked local muscular wasting, and the absence of the reaction of degeneration; the history will reveal the slow and progressive nature of the rachitic process in marked contrast to the suddenness of

poliomyelitis anterior acuta. *Pseudo-hypertrophic paralysis* is liable to be confused with rickets when the feebleness of the limbs causes marked difficulty in the progression of children able to waddle about. This disease shows itself at a later age, and the marked weakness is always associated with obvious enlargement of the calf-muscles; the infraspinatus is much hypertrophied, in association with wasting of the latissimus and of the lower part of the pectoralis major.

Syphilis presents a real difficulty, since the hereditary form of this disease is very often associated with rickets, and many of the changes observed in pure congenital syphilis closely resemble the signs of a simple rachitis. Of these the chief are the natiform skull, cranio-tabes, and anæmia, which many authorities still maintain may exist in syphilis independently of rickets. A careful investigation of the family-history and of the period of the advent of the pathological process usually clears up the diagnosis; but the subject is not of the importance which at first sight belongs to it, since the indications for rachitic treatment are obvious in every case where a syphilitic history is obtainable, and antisiphilitic treatment should proceed obviously in conjunction with the rectification of all dietetic errors.

Little importance need be attached to the systolic murmur often audible over the fontanelles, since this is heard in syphilitic, and even sometimes in healthy children as well as in rachitic ones.

Prognosis.—This is always good; rickets, except for the dangers arising from complications, may be said never to end in a fatal issue. Rickety children are, however, very prone to be easily snuffed out by bronchial disease, especially by the bronchopneumonia occurring in an attack of measles and whooping-cough, owing to the pulmonary embarrassment arising from the thoracic deformity. Convulsions are seldom fatal, but laryngismus stridulus is a frequent cause of death. The ultimate outlook in female children becomes serious owing to severe pelvic deformities. Unless the curving of the long bones is excessive, the outlook as regards permanent deformity and diminution of stature may be said to be good when proper precautions are taken in the management of the case during its acuter stages, many patients entirely recovering without obvious maiming.

Treatment.—The dietetic errors already referred to under the Etiology of the disease afford clear indications for therapeutics. The main difficulty is the discovery of a suitable substitute for the milk of a healthy mother, since the absence of the latter is the most common cause of the mischief. As already pointed out, cow's milk contains enough fat and more than enough proteid for healthy infants, but the presence of

the casein causes digestive disturbances which prevent the assimilation of both fat and proteid. If water be added to bring the proteids up to the standard of normal human milk, the proportion of fat becomes seriously reduced. To overcome this difficulty resort to peptonisation is clearly allowable, but continuous predigestion of the milk will not meet the requirements of the case. The addition of cream and beef-juice is necessary. Proteid may be sometimes safely supplied by raw meat-pulp, varied by the administration of a little entire wheaten flour boiled with the milk. A carefully prepared mutton-broth, with all the vegetables strained out, affords a valuable variety in the dietary of infants of the poorer class. In the children of the better class the disease is often caused by the use of the more expensive artificial patent foods; the substitution of a healthy wet-nurse usually removes all difficulty. At a later stage the dietetic difficulties diminish when soups, egg-yolk, and well-prepared oatmeal-porridge can be used to supplement the diluted cow's milk, whey, and cream. Excess of farinaceous food is as common a mistake as an exclusive diet of over-diluted cow's milk, and the more varied the diet the less danger of dyspeptic troubles at a later stage.

Whilst attention is being directed to the dietary, fresh air, abundant sunlight, and proper warm clothing must be freely provided.

Dyspeptic troubles, when they prove rebellious to changes in the diet or occasional peptonisation of the milk, must be treated upon recognised therapeutic principles, but the less drugging the better; mild purgatives of the castor oil, grey powder, magnesia, and rhubarb class may with advantage be occasionally resorted to. The examination of the stools by careful inspection affords a valuable means of forming a correct estimate of the powers of the patient's assimilation.

An important point too frequently overlooked or neglected is the management of the case as regards the amount of locomotion permitted whilst the bones are still soft. Children should not be allowed even to sit up in bed, except for short periods, as the spinal weakness may cause considerable curvature, and the tibiæ curve from the action of the muscles as the legs are flexed under the body in this position; female children especially should be restricted in their movements on account of the danger of permanent pelvic deformity.

At this stage gentle massage to improve the tone of the weakened muscles and relaxed ligaments is invaluable. In older children who cannot be persuaded to refrain from standing or walking, the application of two long straight splints, applied so that their ends project below the heels, may be serviceable.

Of agents believed to exert a specific action upon the rachitic process there is only one about which there is a consensus of favourable opinion, and this really must be regarded from the standpoint of a food rather than that of a drug—*i.e.* cod-liver oil. It is invaluable at all stages of the disease, even when the child continues to retain its plumpness. Phosphorus has also proved of undoubted value since first recommended by Wegner and Kassowitz; the writer has elsewhere expressed a fear that if pushed it may possibly cause a fixing of the bones in their abnormal position, since, as proved by the experiments of Wegner, it causes great condensation or hardening of the new bone poured out under its influence. It appears, however, to exert a marked beneficial effect over laryngismus, and when prescribed should always be given in combination with cod-liver oil. Not more than $\frac{1}{200}$ to $\frac{1}{100}$ grain should be given thrice daily. Lecithin, protylin, and phytin contain phosphorus in organic combination, and are recommended as substitutes for the crude drug. Phosphates of lime, iron, magnesia, and other mineral phosphates have no action upon the diseased process and in no way can exert any free-phosphorus action in the body.

Suprarenal-gland feeding has been extolled as a specific by Stoeltzner, thymus-gland by Mendel, and thyroid by Ausset.

The anæmia is a clear indication for the administration of iron or small doses of arsenic.

For the permanent deformities of bone, knock-knee, &c., much may be accomplished by surgical measures at a later stage.

RINGWORM, or TINEA

Under this title are included several skin-affections formerly believed to be separate and distinct diseases, but which now are known to be the result of two or more closely allied vegetable parasites—*trichophyton microsporon* and *trichophyton megalo-
sporon*. The different appearances of the affections caused by these organisms are mainly due to the structural peculiarities of the particular tissue which has been invaded by them; hence they are named after their localisation, Ringworm of the scalp, R. of the beard, R. of the skin, and R. of the nails, and will for convenience be described separately.

TINEA TONSURANS

Ringworm of the hairy scalp is a very common disease; and though it is upon the whole the exception to find any degree of permanent baldness caused by it, nevertheless the disease is regarded as a very serious one owing to its tendency

to spread through schools and the great difficulty experienced in eradicating it.

Pathology.—There is still some difference of opinion about the classification of the different ringworm-fungi. The usually accepted division is that maintained by Sabouraud and Crocker, who divide the ringworm parasites into two main groups—the trichophyton microsporon or small-spored fungus, and the trichophyton megalosporon or large-spored form. The prevalence of each form differs widely in different countries. In England the small-spored fungus is the cause of the disease in five-sixths of all cases at least. Colcott Fox states that ‘as with other plants, each country has its own particular ringworm flora, though we may meet occasionally with specimens introduced from other countries.’ Most of the species of the two great families infect domestic animals; the small-spored form is frequently found in the cat, horse, and dog. The writer has witnessed numerous instances in Ireland where ringworm of the body was directly due to infection from cows; and he believes that the large-spored form is usually transmitted from this source, as the scalp was very rarely affected even when the body and face were extensively invaded.

Leslie Roberts reported a few years ago a series of cases of ringworm produced by the fungus from an infected kitten arriving from Egypt. Different species vary greatly in their power of producing irritation, whilst the variations are but slightly marked in different examples of the disease produced by the same species; fresh infection from animals, however, seems to be more virulent than fungi transmitted from one human being to another.

The fungi appear to possess the power of forming ferments, which liquefy the tissue-elements in their immediate neighbourhood, so that the latter are rendered available for nourishment. They produce toxins which excite inflammatory action and disintegrate the epidermis of the hair, so that the fungus finds entrance into the shaft, in which it multiplies in an upward and downward direction both upon its surface and within its substance.

If a stump of affected hair be removed and soaked in liquor potassæ, numerous round spores measuring about $2\ \mu$, or slightly larger, are visible, as a close network, forming a firm sheath surrounding the base of the shaft, whilst a fine mycelial thread may sometimes be recognised in its interior. As a rule, the spores of microsporon are heaped up in zooglœa-masses, the containing mycelium being invisible. The spores of the different species of megalosporon vary considerably in size, being always much larger than microsporon; they measure from 4 to $10\ \mu$ in length. The spores of *trichophyton megalosporon ectothrix* produce ringworm of the beard and of the

nails, and are always, according to Sabouraud, of animal origin. The same fungus is occasionally found producing tinea tonsurans, and is the common cause of ringworm of the body. In the scalp it very usually produces pustulation or kerion and as a rule it is limited to that portion of the hair which is confined in the follicle, and it chiefly affects the outer part of the base of the shaft; hence its name—ectothrix. *Megalosporon endothrix* is another species of the large-spored fungi; it spreads by the formation of a sporulated mycelium, which extends from the root of the shaft along its entire length, keeping always in the interior of its substance. The spores of megalosporon are always contained in distinct mycelium-filaments. The large and small spored parasites are never found together in the same cutaneous lesion.

Etiology.—The common form of tinea tonsurans, which is the result of the invasion of the small-spored fungus just described, is a markedly contagious and obstinate disease, spread by transmission of the spores from an infected scalp through combs, brushes, towels, caps, &c., or by direct contact, or by the spores finding their way from the horse. Less frequently it arises from a patch of body-ringworm, produced in the first instance by any of these methods of transmission. The disease is practically confined to children; very rarely has it been known to commence after fifteen. The few cases of tinea tonsurans recorded in adults are examples of the rarer large-spored species of fungus. The disease may begin in infancy, and if neglected may continue long after puberty. Boys are affected as frequently as girls, and the disease attacks the healthiest children, though it often appears to flourish more luxuriantly in the under-fed and unhealthy. It is stated also to be more frequently met with in fair-haired children; but, as pointed out by Crocker, fair hair is more common than dark in Britain. As age advances the hair-shafts and follicles in the scalp-region become more resistant to the fungus, though the hairs of the beard retain their vulnerability to the large-spored species.

Symptoms.—As first seen, the disease appears on the scalp as a small reddish circular spot or patch, covered with fine whitish scales, resembling a circumscribed area of dry seborrhœa. The patch increases in size gradually by an even peripheral expansion, whilst maintaining its perfectly round form and sharply defined border, surrounded by a narrow margin of erythematous redness. The hairs are broken short, and the irregular, bent, or twisted stumps are observed sticking out from the patch like pins in a pin-cushion. These stumps are dull, dry, and withered-looking, and, though not longer than a quarter of an inch, and brittle, they readily are pulled out by a pair of forceps. Occasionally neighbouring patches join together so that gyrate outlines are produced,

which may include between them a pretty extensive area of the scalp, though the individual patches which keep discrete and rounded seldom exceed two inches in diameter.

The stumps when pulled out are so characteristic, even to the naked eye, that the disease can hardly be mistaken for anything else; when closely looked at, the proximal end of the stump may be observed to be covered with a light-coloured layer or sheath, which is seen under the microscope to be made up entirely of spores. The amount of irritation or inflammation present in the patch varies considerably; as a rule, it is confined to a few pustules here and there visible when the scales are removed, but the condition known as *Kerion* is sometimes observed. Though this has been repeatedly stated to be characteristic of megalosporon, it is unquestionably sometimes in its typical condition produced by the small-spored fungus. The parasite excites a pustular inflammation in the hair-follicles of the diseased patch which causes it to stand out above the level of the healthy skin; the patch feels boggy, the stunted hairs, if not already shed, are quite loose, and from the mouth of each empty follicle a drop of glutinous pus may be squeezed out which may contain numerous pus-cocci. It is probable that kerion is more likely to occur when the fungus has reached the scalp by direct infection from the skin of an animal than when the disease has been communicated from the affected scalp of another child. The type of ringworm in which kerion forms is liable to lead to considerable permanent baldness, but, according to Morris, sloughing never occurs.

Old patches of *tinea tonsurans* tend to become very scaly and somewhat thickened, and the stumps of the stunted hairs are often so short as to cause an appearance of complete baldness in the patch. This bald appearance is well marked in the megalosporon endothrix ringworm, and the patch is usually a large somewhat irregularly shaped one formed by the coalescence of several round spots. There is usually very little scaliness, so that the bald area looks smooth, and when closely investigated numbers of black dots are observed; these are found to be the short stumps of the hairs, which are liable to break off close to the skin-level.

The progress of the disease is almost without termination if left to itself; many years may often elapse before its final eradication, even in the face of moderately active treatment. The natural tendency is for the fungus to fail in its reproduction, or even to perish at some spots when all the hairs have dropped out, whilst at another the spores may be actively growing. At this chronic stage spiral or twisted stumps may be here and there found disseminated amongst the weak, lustreless, attenuated hair-shafts, and much general scaliness prevails. As long as any of the characteristic broken

stumps can be still found the disease is certain to break out afresh upon the suspension of active treatment.

Diagnosis.—The discovery of loss of hair in children, with marked scaliness of the scalp occurring in round patches, in which irregularly projecting broken stumps are seen, leaves little room for doubt; the microscope settles the diagnosis in all cases easily. There need be little said about the differential diagnosis of the different forms of ringworm produced by the various species of the small and large spored fungi, as the subject is one of little practical interest and has no bearing upon treatment. Sometimes in infants and in very young fair children with delicate fine hair the first appearance of the disease may be in the rings which characterise ringworm of the smooth parts of the body, but the rings soon fill up and become round patches.

Old neglected cases, where the disease is scattered through the scalp, may be mistaken for ordinary *dandriff*, but the uniformly diffused distribution of this variety of seborrhœa is characteristic. The ringworm-patches, though numerous, show well-defined borders, and they are almost always found in younger patients than those in whom seborrhœa is present. The stumps, when carefully sought for, are always to be found amongst the scaly patches.

Eczema often presents a real difficulty under circumstances like the following: The physician sees a case that has been under active treatment perhaps for a long time, even after the fungus has been entirely removed. The remedies employed for the destruction of the disease may have set up a severe traumatic eczema, so that the case may be supposed to have been an eczematous one from the commencement. Upon the suspension of the use of the irritant the inflammatory action subsides; but a careful search with a large convex glass should never be omitted, and the forceps should be employed to test the looseness of any suspicious ill-nourished hair-shafts, even when no broken stumps can be detected. *Psoriasis* is not likely to be mistaken, even when there are no patches on the knees and elbows, because the silvery scales upon the scalp are piled up in crusts in quantity far beyond that observed in ringworm; the nutrition of the hair is but slightly interfered with, and broken stumps are never seen.

Favus is differentiated by its primrose-coloured and cupped discs with mousey odour, and the hairs show no broken stubble or stumps.

Prognosis.—The disease is always curable, but most intractable when not taken early. The outlook as regards duration is therefore a matter chiefly of the length of start which has been given to the fungus before the application of remedies. It may in long-standing cases require many months, or a couple of years to effect a cure.

Treatment.—A considerable change has come over the views about the therapeutics of *tinea tonsurans* within the past two or three years, and in the transition-stage much confusion has arisen. After all, the old principles remain, only details being altered. The hair about and around the diseased patches should be clipped as close as possible with a pair of sharp-pointed scissors. After this, epilation when practicable should be resorted to, each hair in the diseased area being pulled out by a suitable epilation-forceps. The process is painful and tedious, and in the case of very young children therefore almost out of the question. After the complete removal of all diseased hairs a parasiticidal ointment should be thoroughly rubbed in. The formulæ for these are without limit, but it may be safely said that it is a matter of little importance whether strong iodine, a mercurial salt, copper, chrysarobin, salicylic acid, creosote, formalin, or other antiseptic drug be selected. Of infinitely more importance are the repeated epilations and the thoroughness and unflagging care exercised in the application of the remedy selected. By constantly changing this the physician never becomes real master of the use of any one agent. The writer believes that for all routine work the ointment of iodide of sulphur is the best, once the physician has made himself acquainted with the amount of irritation which it usually produces. A detailed list of the remedies used by different dermatologists will be found in the last edition of the author's 'Dictionary of Treatment,' and need not be here reproduced.

The main element in the success of all treatment is the introduction of the germ-destroying agent into the hair-follicles, and this can only be effected after epilation. The forceps fails to remove the stumps when these get thoroughly permeated by the fungus, as they break off often with the slightest traction; hence the success of the old croton-oil and blistering methods, which effect epilation with or without the production of kerion. These methods, however, will probably soon be only of historic interest, since the scientific application of the X-rays by Sabouraud effects epilation in a manner superior to all other methods. The rays have no destructive action upon the fungus, but they permit of the effective use of antiseptic applications in a way hitherto impossible by the older methods. Sabouraud states that by their use the old average of two years' treatment is now reduced to three months. In a week after the exposures have been commenced the shafts begin to loosen, and the hairs drop out all over the affected area in another ten days without pain or inflammation, and do not grow again for at least three months. Oil of cade ointment (two parts of the oil to five of lanoline) is well rubbed-in nightly, whilst the entire scalp is washed every morning, and painted afterwards with

weak tincture of iodine. The technique is described in 'La Presse Médicale,' December 28, 1904.

TINEA CIRCINATA

Also known as Herpes Circinata, is the name given to ringworm of the glabrous skin. The small-spored fungus is the most common cause, megalosporon producing only a variable minority of the cases. The disease appears usually in a distinct ring, developing from a small pinkish, round, raised, scaly patch, which spreads peripherally whilst it resolves in the centre. The ring may reach to the dimensions of two inches in diameter, and by the coalescence of neighbouring rings festoon-patterns may be formed like those seen in some varieties of psoriasis. When the disease attacks the arm-pit and inner aspect of the upper part of the thigh the fungus grows luxuriantly in the moist warmth, forming large gyrate patches, with raised eczematous thickened borders. This form is nearly always caused by the large-spored fungus; it is known as *Tinea cruris* or *T. axillaris*, and is frequent in warm climates. It is sometimes described in Britain under the title of *Eczema marginatum* when it spreads over the lower part of the trunk and buttocks.

When ringworm of the body occurs in solid round patches which do not resolve in the centre, these are generally much smaller than the rings, and remain scaly throughout, with well-defined margins. It is usual to find, however, typical rings in their vicinity. As in the scalp, there is generally no itching and often no pustulation or other inflammatory action; but the large-spored fungus, which usually forms more extensive rings, is not rarely associated with considerable pustulation or herpetic vesicles around its raised and thickened margins. These cases can generally be traced to direct infection from domestic animals or cattle. Rarely does kerion appear, but, as in the scalp, it may occur even when the microsporon is the cause. Concentric rings are very occasionally observed.

The disease may attack any portion of the trunk, extremities, or face; most frequently the rings are observed only upon the exposed parts; they are never, except by accident, symmetrical, and they vary obviously with their age.

Ringworm of the body is easily recognised by the above characters, which distinguish it from all other cutaneous affections, except when seen after the application of irritants which have caused considerable eczematous inflammation in the surrounding healthy skin. The fungus is readily identified by the microscope in all doubtful cases.

Treatment.—The disease is cured with ease and certainty by a few applications of any antiseptic solution or ointment in its early stages. The old B.P. liniment of iodine is usually

very reliable ; but in examples of the affection caused by direct infection from animals the fungus is usually more virulent and tenacious of life, and the iodide of sulphur ointment, chrysarobin, or perchloride of mercury may be necessary. In infants and young subjects with very delicate skins a solution of hyposulphite of soda (1 in 10) is often efficacious, or the *tincture* of iodine may be painted on after removal of the scales.

TINEA SYCOSIS, RINGWORM OF THE BEARD, or BARBER'S ITCH shows itself as a pustular folliculitis, nearly always caused by trichophyton megalosporon ectothrix of animal origin, conveyed by the lathering brush used before shaving. It almost invariably occurs in those who are shaved by barbers, but the writer once saw it in a patient who never shaved, the disease being due to infection from a patch upon the neck, probably transmitted by scratching.

Symptoms.—The first evidence of the disease is often a severe itching confined to a small raised patch of scaly red skin, situated usually upon the chin. Its borders are thickened, being formed of irritable papules, vesicles, or pustules, and, like ringworm of the body, it may be distinctly ringed, with a normal centre, or may remain as a solid red scaly patch throughout. With the irritation produced by constant shaving the tendency to pustulation and the formation of kerion increases, and the suppuration is often intensified by the introduction of pyogenic cocci.

As the disease spreads a considerable area of the hairy portion of the face becomes invaded ; large, raised, brawny or boggy, red, and irritable patches or lumps form, which discharge pus freely, and as shaving becomes impossible these are seen to be covered with the short stubble which has grown since the last cutting. The stumps are apparently normal in appearance, but they are always quite loose ; each is surrounded by a small pustule, and can be readily plucked out without causing pain. The patches are the seat of a burning itch, and even throbbing may be experienced, owing to the spreading of the inflammatory swelling caused by the irritation of the fungus as it develops in the interior of the large deep hair-follicles.

The disease is most intractable and pursues a chronic course for years, unless treated actively ; owing to the severity of the suppurative process permanent scarring and loss of hair may occur over a considerable area on the chin or side of the face.

Diagnosis.—The disease must be differentiated from the so-called *non-parasitic sycosis* which is caused by *staphylococcus albus et aureus*, and hence termed by Unna Coccogenic sycosis. This affection commences as a peri-folliculitis, which,

spreading inwards, converts the follicle into a minute abscess. The symptoms resemble those of tinea sycosis, but the progress is never so rapid in its early stages and the hairs for a long time do not loosen. Even after there is considerable pustulation the shafts can only be pulled out with force, causing much suffering to the patient, and they are found to be normal in appearance and not brittle; the root-sheaths are swollen; whilst in tinea sycosis the sheath is commonly absent. The microscope settles the diagnosis in all cases of tinea, but the fungus is often only to be detected in those few stumps where the root-sheath remains attached to the base of the shaft.

Eczema is differentiated by the history of a serous discharge, by the discovery of similar patches somewhere upon the smooth parts of the face, and by the fact that the hairs never become loose or fall out.

Treatment.—This is essentially similar to that of tinea tonsurans, the chief point being the complete removal of every affected hair by epilation with the forceps, or preferably by the discreet use of the X-rays, after which any mild parasiticide ointment must be rubbed in carefully. Carbolic acid or creosote (1 to 6) with lard is convenient and efficacious. Shaving should be discontinued, the beard being clipped daily with sharp-pointed scissors. Obstinate cases yield to a weak iodide of sulphur ointment (1 in 30). When poulticing is necessary for the removal of crusts, a compress of warm boric acid under oiled silk answers every purpose.

TINEA UNGUIUM, or RINGWORM OF THE NAILS

This, known also as Onychomycosis, is usually secondary to ringworm of the body or head, and is generally caused by the large-spored ectothrix fungus. The disease is probably the result of scratching. Whether the parasite gains entrance at the free border or by the side of the nail, the results ultimately are that inflammation of the matrix is excited and the substance of the nail becomes thickened or infiltrated and brittle, splits and chips, and loses its lustre; ridges, grooves, and striæ deform it, and ultimately it becomes of a dirty brown or black. The disease may last for many years, and obstinately resists treatment.

The nail, after being scraped and brushed with liquor potassæ and water (equal parts), should be enveloped with a weak perchloride of mercury solution (1 in 100), applied on lint and covered with the finger of an indiarubber glove.

ROSACEA

This is a chronic erythematous inflammation of the skin, generally limited to the region of the nose and to the cheeks, involving the sebaceous glands and eventuating in dilatation of the superficial local blood-vessels and in irregular thickening of the skin.

Etiology and Pathology.—The condition in its milder forms is more commonly seen in women than in men; in the writer's experience, the most advanced and disfiguring varieties of the affection are always met with in males. Its first appearance is usually at about the period of thirty to thirty-five years, appearing as a fleeting erythema following exposure to variations of temperature, especially in women the subjects of ovarian or uterine irritation and in individuals suffering from dyspepsia or chronic gastric disturbances. In women the erythematous condition often becomes established permanently about the climacteric period. Morris points out that the simplest type of rosacea is to be seen in the transient grog-blossoms of the habitual drunkard. These are the result of a vaso-motor neurosis called into existence by reflex irritation; at first only temporary erythematous redness occurs, but this ultimately ends in permanent dilatation of the superficial blood-vessels, associated often with inflammation in and around the sebaceous follicles. The chronic irritation terminates in proliferation of the connective tissue, causing, especially in the case of male alcoholics, great hypertrophic thickening and the formation of irregular excrescences or tumours. The affection in this final hypertrophic stage is known as *rhinophyma*, in which often pendulous growths, lobulated tumours, or tomato-like excrescences cause remarkable unsightliness or deformities, which Hans Hebra regarded as a different affection from ordinary rosacea. The implication of the sebaceous glands, so often present, led to the old name for the affection—*acne rosacea*; but the presence of acne is not a necessary element in the disease.

Crocker, along with most dermatologists, accepts the pathogeny of the affection as that of a primary vaso-motor reflex neurosis; whilst Unna maintains that the condition should be regarded as a modified form of seborrhœa. It is interesting to note in this connection that Lloyd has recently drawn attention to the fact that the source of reflex irritation is sometimes to be discovered in the nasal cavity itself in the form of polypi, enlarged turbinates, hypertrophic rhinitis, &c. It is unfortunate for the victim of rosacea, or of its most aggravated and advanced variety—*rhinophyma*—that the popular idea of its etiology is associated with alcoholic excesses and gluttony; some of the worst cases met

with occur in the most abstemious subjects, who never taste alcohol.

Symptoms.—The symptoms and signs of the condition have been already incidentally described in the remarks made regarding its pathological anatomy. The erythema, at first transient or fleeting, and always aggravated by exposure to cold winds and dietetic errors, becomes gradually established as a permanent redness, in which numerous congeries of dilated blood-vessels appear over the lower part and wings of the nose. Then follows hypertrophic thickening, in which acne papules and pustules here and there appear; more or less encroachment of the redness and dilatation of the superficial vessels are usually observed upon the neighbouring skin of the cheeks. The advanced hypertrophic lobulation and tumour-formation which go to make up rhinophyma is still less liable to be mistaken for any other affection.

Diagnosis.—From *acne vulgaris* the disease is differentiated by the age of the patient, the history of the onset commencing with diffuse flushing and often dyspeptic troubles; there are no black-heads or comedones, and the follicular inflammation and dilatation of the venules only appear at a late stage and not at the commencement of the affection. This latter point readily distinguishes the condition, even when retention of the sebaceous matter and free pustulation have occurred similar to what is seen in acne; moreover, the symmetrical localisation of the affection on the nose and corresponding level of the cheeks and the absence of inflamed sebaceous follicles upon the shoulders or elsewhere are distinctive.

Lupus erythematosus is differentiated by the scaly condition of its raised borders, by the presence of atrophic scarring, and by the butterfly-distribution. *Tertiary syphilitic* eruptions are non-symmetrical, show a tendency to break down or ulcerate and scab, and always are found associated with other late manifestations of the disease, and, unless by rare coincidence, are not found in connection with obviously dilated venules.

Prognosis.—The tendency is towards chronicity and permanence, unless active and persistent treatment be carried out.

Treatment.—Mild cases of rosacea may recover completely when the removal of the cause is effected, the dyspeptic form being speedily amenable to treatment when taken at the stage of erythematous redness. The disease in its established form is very intractable. After the supervention of inflammation of the follicles mild local treatment suitable in acne is indicated. Where hypertrophic pendulous growths have formed, surgical measures are necessary to remove the deformity. The dilated vessels can be often obliterated by multiple scarification or electrolysis, after which adrenalin

should be locally applied ; the appearance of the patient may be thus greatly improved without removal of tissue. Leredde attaches great value to vegetarianism, and in all cases alcohol in every form must be prohibited ; excellent results are often obtainable by ichthyol internally in 5-15 min. doses.

ROSEOLA

This term is generally used as a synonym for Rose-Rash, Rubella, or Rötheln, described under 'Measles (German).' Roscola, unfortunately, is used in a somewhat loose sense by dermatologists to include any mild form of non-exudative erythema with diffuse or general distribution, the result of internal causes. One idiopathic variety of this described by Crocker as *Erythema Roseola* and another symptomatic variety which he calls *Erythema Scarlatiniiforme* answer closely to the affection described by Clement Dukes as the 'Fourth Disease,' hitherto regarded as a form of German measles, only that these affections are not contagious and there is no enlargement of the lymphatic glands, which Dukes maintains always to be present in the 'fourth disease.' The chief importance of erythema scarlatiniiforme lies in its liability to be confused with scarlatina and rötheln. From the former disease it is differentiated by the early stage at which desquamation occurs. Though the eruption may last for two or three weeks, desquamation may be often observed upon the second or third day while the roscolar rash is still bright. Sound patches of normal skin may be usually found between the diffused areas of the scarlatiniiform rash, which, moreover, sometimes remains confined to the trunk. There is reddening of the fauces, but no swelling, and though a strawberry type of tongue is often present, it never assumes the distinctive form seen in scarlatina, and the temperature seldom rises beyond the febrile range. A form of the affection is recognised in which frequent recurrences take place, sometimes at yearly intervals, in adults, always followed by more or less extensive desquamation.

The term 'roseola' is also applied to the various eruptions of the exanthemata, syphilis, beri-beri, pellagra, cholera, and to many of the drug-eruptions.

RÖTHELN

This is described under 'Measles (German),' p. 979.

SCABIES, or ITCH

This is a contagious disease caused by an animal parasite whose bites, owing to the inoculation of an irritating virus, produce severe itching, leading to the formation of multiform skin-eruptions through scratching, and the production of characteristic burrows by the female in the act of laying her eggs.

Etiology.—The disease is very common and widespread, affects individuals at all ages from infancy to senility, and is found most frequently in persons of uncleanly habits. It is conveyed by close and prolonged personal contact, or through the wearing of infested garments, gloves, bed-clothing, &c.

The parasite—*Acarus scabiei* or *Sarcoptes hominis*—belongs to the class *Arachnidæ*; in its adult condition it possesses eight legs, which excludes it from being regarded as an insect. The male is considerably smaller than the female and is seldom captured; he may be recognised by the suckers attached to the last pair of hind-legs, which, moreover, do not carry bristles. The female, when impaled upon the point of a needle thrust into the blind end of a burrow, appears as a minute white globular body, measuring $\cdot 3$ mm. or about the $\frac{1}{80}$ of an inch; under the microscope its breadth is seen to equal its length. The two anterior pairs of legs have small suckers attached, whilst large setæ project from the extremities of the two hinder pairs. The general configuration of the parasite is bug-shaped; its convex bristly back exhibits transverse striæ and serrated lines. It possesses sharp-pointed mandibles, which, according to Bourguignon, it employs for puncturing the skin of its host, after which it squeezes the blood into its mouth. The irritating nature of its salivary secretion, and not the mechanical effect of its bristles, is believed by Mégnin to cause the intense itching.

The life-history of the acarus must be known in order to understand the symptoms to which it gives rise and the best methods of effecting its eradication from the body. Only the female burrows, the male acarus keeping upon the surface of the skin, sheltered under the crusts of the secondary lesions. After impregnation, with the view of depositing her eggs she penetrates the horny layer of the epidermis, and, forming a tunnel or cuniculus, she lays one or two eggs daily at different points in the burrow, which may measure more than half an inch in length by the time her fifty eggs have been deposited. At the blind end of the burrow the acarus dies after laying her last egg. The entire tunnelling operation occupies more than a month; the eggs are hatched sometimes within a week, and the larvæ at the free end of the burrow are well developed whilst the ova are being still deposited at the blind

end. The larvæ either make their way out by piercing the epidermis or emerging through the original opening. Upon their first appearance they possess but six legs ; after forming a superficial burrow later on, the larvæ moult there two or three times, and finally emerge as perfectly formed males or females, to commence the sexual stage of their life-cycle. The acarus appears to feed and to perform its tunnelling operations at night.

Symptoms.—The situation of the tunnels seems to be determined by the nature of the skin ; the acarus selects the soft delicate folds between the fingers and the creases on the front of the wrists, the inside of the thighs, front of the abdomen, genital region, axillæ, and groins. The face and scalp are never invaded except in infants, in whom also a favourite site is the feet ; the back, shoulders, and upper part of the chest are only affected, as a rule, in cases of long standing.

The cuniculus or burrow may be detected by the sinuous or irregular outline of its trail, which in length varies considerably ; the majority of them do not exceed one-fourth to one-half an inch, but twice this length of tunnel is not rare. The track is usually a dirty black or brown in persons who do not wash frequently. By using a hand-lens the blind end of the burrow may be detected, with the female implanted in it as a minute shining speck, and backwards its wavy outline may be traced as a black line, often broken by the presence of a pustule or vesicle, the trail ending on the surface of the skin at the original opening, with its dirty frayed-out edges.

The occupation of the patient influences the site upon which the burrows are to be found. In those whose hands are being continually immersed in water, as in the case of washerwomen, the burrows are destroyed as soon as they are formed, and are consequently absent on the hands, wrists, and fore-arms. For similar reasons they are not to be found upon the hands of brickmakers and bricklayers. In tailors and cobblers the burrows and other lesions are usually well-marked on the buttocks.

Multiform lesions are the result of the scratching indulged in to relieve the intense pruritus caused by the bite of the acarus. Papules arise whose tops are soon mowed down by the finger-nails, and these become tipped with crusts ; vesicles and pustules also result from the traumatic dermatitis ; whilst in other subjects the inflammatory process tends towards the clinical picture of an eczema or an urticaria with large wheals. The most common general result, as a rule, is the production of an eczema impetiginoides from the presence of pus-cocci. These infect the finger-nails and are sown in the parallel erythematous scratch-abrasions, so that pustules may be found at a considerable distance from any burrows.

The site of the multiform eruption, however, in the main corresponds with the region in which the burrows are very numerous; hence it is most common between the fingers and about the wrists, on the inside of the thighs and front of the abdomen, &c. In infants the face and scalp are often covered with crusts, but the hands and feet seldom escape, and the buttocks are generally involved from contact with the hands of the infected nurse or mother.

If not successfully treated, the disease may continue indefinitely till the entire cutaneous surface is covered with the multiform lesions.

Diagnosis.—The main points have been emphasised in the above brief description. The recognition of the disease is of great importance owing to its contagious nature, and many professional reputations have been shattered by failure in recognising the nature of the eruption till all the children in a large school had become infected. The discovery of a single acarus in her burrow settles the diagnosis; but the writer can emphasise the difficulty often experienced in finding the parasite when he was absolutely certain about the disease from observing the mixed nature of the lesions. An entire burrow may be snipped out by using a pair of fine-pointed sharp scissors, and the larvæ and eggs recognised under the microscope.

Eczema of the hands should be differentiated from scabies by the grouping together of the minute vesicles which are characteristic of eczema in this region. In scabies the vesicles are larger and usually discrete, being scattered about in an irregular manner—a characteristic which is almost pathognomonic of itch. It must be always remembered that the parasite is never to be found in the contents of a pustule or vesicle, but at the blind end of the burrow.

The greatest difficulty in the diagnostic problem resembles that so frequently to be met with in *tinea tonsurans*. The opinion of the physician is sought at a stage after the injudicious use of household remedies which have produced a severe dermatitis, the clinical picture being that of an acute and severe eczema. A careful examination fails to detect burrows, but if the entire body-surface be inspected and the general distribution of the lesions before treatment be investigated, a mistake is very unlikely to happen. In the writer's experience, mistakes usually arise from confining the examination of the cutaneous surface to a small area of the body; when possible, the entire skin should be uncovered and a general survey made. The fact that more than one member of the family or household is affected should always arouse suspicion.

Urticaria may be the most prominent lesion in some children, and the presence of the acarus may not be suspected. The

situation of the wheals should arouse suspicion, after which the discovery of burrows follows.

Treatment.—The disease is easily cured. The burrows must be opened up by prolonged immersion of the body in a warm bath, followed by thorough scrubbing with a suitable flesh-brush and abundance of soft-soap. After rapid drying the B.P. unguentum sulphuris should be rubbed in for half an hour. Two, three, or four such applications suffice to cure the disease, even in long-standing cases. Much care, however, will be necessary in dealing with patients whose skins are delicate or in whom extensive dermatitis has already occurred. The number of parasiticide applications is endless; a list will be found in the author's 'Dictionary of Treatment' (4th ed.), pp. 864, 865, where the rapid, cleanly, and efficacious method of treating the disease by the use of a home-made solution of pentasulphide of calcium is detailed.

Thorough disinfection of all clothing must be carried out by baking in an oven or ordinary disinfecting chamber.

SCARLATINA

This disease, also known as Scarlet Fever, is an infectious fever, characterised by the presence of sore-throat and a bright scarlet punctiform eruption, followed by desquamation and exhibiting a marked tendency towards certain sequelæ.

Etiology.—The disease is widespread, being found in all climates, but very dry and hot regions suffer much less than the more temperate ones; in these latter the disease seems to be usually present in sporadic form, occasionally breaking out into more or less severe epidemics. In India, China, and Egypt scarlatina is comparatively rare, whilst in Japan it is stated to be unknown. Few, if any, epidemic diseases show such marked variations in their intensity and mortality; but a consensus of opinion shows that within the last thirty to fifty years a much milder type is being met with than was common up to the middle of the last century.

The disease always is the result of contagion; the specific organism is conveyed by contact of the healthy with those suffering from it. The breath, saliva, nasal, and pharyngeal secretions undoubtedly contain the virus, which also exists in the skin and is readily conveyed by fomites; it adheres to clothing, toys, books, furniture, and every article in the sick-room. It may be readily conveyed by healthy subjects who have come into but slight contact with the sick, and numerous instances of its transmission have been traced to cats, dogs, birds, and other pets which have been the carriers of the virus upon their fur or feathers without themselves being infected. There still exists a very incorrect estimate of the

intensity of the poison in the desquamated epithelium. There cannot be a doubt that the disease is most highly contagious in the stage of eruption, and that the clothing worn by the patient at this early stage before he takes to his bed may retain the germs of the disease in a high state of potential activity for long periods. Some authorities deny the infectivity of the desquamated epithelium in the late stage of the disease, so that now in some fever-hospitals the scarlatina-patient is permitted to go home in the last couple of weeks before the shedding process is completed. This seems, with our present lack of knowledge of the life-history of the specific microbe, to be risky, but it marks a rebound from the old idea of the virulence of the dead epithelial flakes. There is practically no danger in the disease being transmitted by the desquamated epidermis when antiseptic inunctions have been resorted to from the beginning. The urine and fæces probably contain the virus, but since it cannot be regarded as a water-borne disease the spread of infection in this manner may be ignored; the exhalations from the nose, throat, and respiratory tract apparently afford the most rational explanation of the spread of the disease.

Infection by a contaminated milk-supply has been clearly proven, and this fluid may be infected by the germs given off in the sick-room, or it may become impregnated with them owing to the milch-cow suffering from the disease.

Of the predisposing etiological factors some are of the greatest importance. Age is a powerful influence: the great majority of all cases occur before the tenth year, but infants under one year are very slow to catch the disease. Many individuals escape entirely if they happen not to have come in contact with infected subjects during childhood, as susceptibility certainly begins to fade before puberty sets in. Hence the importance of rigid isolation; but of even greater moment is the fact that the severity of the disease is greatly influenced by the early age of the patient, the attacks, as a rule, being progressively milder after the seventh or tenth year. The longer a child can be shielded from exposure, therefore, the better, especially when the next factor is taken into account. Idiosyncrasy as regards susceptibility is a remarkable feature in some families; numerous instances are on record where large families have been entirely wiped out by the disease, whilst the victims in surrounding families escaped with mild attacks. This family-susceptibility, however, appears often to follow the general law of becoming less marked as age advances, so that often only the youngest members succumb to the attack, the older ones escaping with their lives or only suffering to a comparatively mild extent.

One attack as a rule confers immunity; the great majority of instances of second and third attacks are examples of errors in

the diagnosis of the primary seizure, which may have been an attack of r  theln, measles, or the so-called 'fourth disease.' This error also explains the curious relapses which are said to occur in the progress of an attack; these are due to the supervention of one of these last-named diseases.

Seasonal influences are observable; the disease occurs much more frequently in the autumn, and assumes then a more virulent type as a rule than when it appears in the spring months. Sex plays little part in the incidence, males and females suffering about equally; but Dukes states that a fatal termination is more common in males.

Bacteriology.—The specific organism which causes scarlatina cannot yet be said to have been conclusively demonstrated. Gordon maintains that a micro-organism allied to, but distinctly different from, streptococcus pyogenes is the specific factor; to this coccus he gives the name *Streptococcus scarlatin  * or *S. conglomeratus*. It is always found in the mucus on the tonsils or pharynx of scarlatina patients and often in the blood and serous effusions. Mallory found a parasite like that of malaria in the superficial layer of the skin. Many observers affirm that the disease is the result of a modified or mixed streptococcic invasion. In connection with this latter view the recent researches of C. J. Lewis are important; he examined the secretion from the purulent otorrh  a and rhinitis of scarlatina in over seventy cases. In otitis the pneumococcus, streptococcus pyogenes, and a diphtheritic bacillus were usually present, the pneumococcus being found in more than half the cases. In the nasal discharge streptococci, staphylococci, and pneumococci were also found, and sometimes bacilli of the diphtheria-group. Whatever form of specific organism is to be ultimately proved as the cause of scarlatina, it is therefore obvious that the complications are frequently, if not always, the result of a mixed infection, and it is noteworthy that the diphtheria-bacillus plays a very important *r  le* in the suppurative condition of the ear and nose. Hunter has drawn attention to the fact that the septic complications of scarlatina are greatly increased or intensified by a previous condition of oral sepsis; but Goodall insists that the most severe complications of the disease may supervene where there are no evidences of oral sepsis whatever.

Morbid Anatomy.—The skin, throat, and kidney are the common sites of the lesions, but none of these can claim to be of a distinctly specific type. The blood has been found to contain streptococci, but these may be obviously the result of secondary infection, even when found in pure culture.

The eruption is absent after death, but the proliferation of the cells in the rete-layer may be evident; these, however, are only what might be expected in any severe form of

cutaneous hyperæmia. The same remark applies to the hyperæmic condition of the throat, which, if it has been severe, leads to tonsillar œdema and purulent deposits, with the formation of ulcers or sloughs and involvement of the cervical lymphatic glands, the latter becoming invaded by streptococci.

In the kidney the changes are more obvious, but cannot be regarded as specific. The organ is deeply congested and its capsule strips off readily; an acute inflammation of the epithelial lining of the Malpighian corpuscle and proliferation of the cells covering the capillary tuft are present, and are usually described as a 'scarlatinal glomerulitis.' The parenchyma is usually also affected, the cells lining the convoluted tubes showing changes of the type met with in acute nephritis from other causes. The ultimate renal changes found in the bodies of subjects succumbing at a later date from the renal sequela are not to be distinguished from those observed in the large white and in the small contracted kidney.

The hyperæmia and hyperplasia observed in the spleen, liver, salivary glands, and pancreas resemble those found in other acute inflammatory infective processes. Changes in the endocardium, pericardium, and serous membranes are common even when evidence of joint-implication has been wanting during life; these are of the usual inflammatory type, resembling the condition of the heart found in acute rheumatism, and sometimes degenerative changes may be detected in the myocardium itself.

Symptoms.—These vary in severity more than in most other diseases, some patients passing through the attack without exhibiting any evidence of illness till the advent of severe renal inflammation causes symptoms of acute uræmia. A moderately severe attack of the disease in its commonest form will be described, and the deviations from it noted incidentally.

Incubation.—This may be accepted, according to Dukes, as never exceeding seven days; it may be as short as twenty-four hours. Usually it is of about three days' duration and devoid of symptoms of any kind.

Invasion.—This is usually more or less sudden or abrupt, with a feeling of malaise, headache, sometimes chilliness and slight pains in the limbs. Vomiting is a very common, and therefore a diagnostic, symptom in all severe cases, and a convulsion in young children very often is the first indication that anything is wrong. Thirst is complained of early, and in a few hours pain in swallowing supervenes; often this latter is the first symptom to attract attention. Fever is present; the temperature rises rapidly and may reach 103° or more within the first twelve hours; the skin is pungent, feeling as if burning to the touch of the observer; and the pulse is always markedly accelerated, even when the fever

is of slight degree. The tongue shows a white fur, sometimes even before the rash appears; but catarrhal symptoms are absent and cough does not occur. The eyes are bright and the face somewhat flushed.

Eruption.—When all the above symptoms of the invasion are present the attack may be predicted to be a smart one. The rash appears twenty-four hours after the initial symptoms of sore-throat and fever. Its first appearance is usually upon the neck and upper part of the chest, but the face is certainly often invaded early. It spreads rapidly downwards, covering the entire cutaneous surface save the palms of the hands and soles of the feet.

The rash when fully out is a bright-red colour, sometimes so vivid as to suggest the tint of a boiled lobster; in very severe cases the redness is tinged with slight lividity, so that the eruption is often described as dusky. When seen at the very commencement the rash will be found to consist of numerous minute red points or punctate papules, separated from each other by normal-coloured skin. These usually remain discrete about the groins and axillæ. As the eruption blossoms out to its typical state these red points become more numerous, and each is surrounded by skin of a hyperæmic or erythematous redness, so that the entire rash presents the appearance of a bright diffuse sheet of scarlet. Though smooth to the touch at first, the minute elevations may be distinguished by a lens; at a later stage the skin feels rough, especially upon the outside of the limbs.

The erythematous nature of the eruption is seen by making slight pressure with the finger, which causes the redness transiently to disappear, and by drawing the finger-nail across any part of the skin the track is marked by a white line. Occasionally true minute petechiæ may be observed in severe cases; these do not disappear on pressure. Sudamina often develop, and a fine miliary eruption may blend with the diffuse redness; the minute vesicles show milky opalescence. The eruption attains its perfect development within about seventy-two hours from its first appearance—*i.e.* upon the fourth evening after the first complaint of sore-throat. From this period it commences to fade, the scarlet becoming more dusky and tending towards a dirty bluish-red. When the eruption is fully out some œdema of the skin is present, so that the eyelids, back of the hands, and feet appear swollen, and there is usually a considerable degree of itching present.

Though the palms and soles may be said to remain free from the characteristic punctate eruption, they usually show a faint diffuse redness, like the flush which appears on the cheek, contrasting with the normal pale colour of the skin in the zone round the mouth whilst the eruption may be well

marked upon the forehead, temples, and behind the ears. The intensity of the rash is usually in proportion to the severity of the attack, but in the somewhat rare malignant type of the disease the rash may never develop its characteristic scarlet hue, remaining purple or livid till death. In very mild cases the rash may be so slight as to escape observation; it may even be entirely absent in rare cases, but nevertheless desquamation follows on the palms and soles at a delayed period.

Desquamation commences after the fading of the eruption, which usually disappears entirely within six or eight days; but streaks of hyperæmic redness remain for some days in the hollows behind the knees, in the skin on the front of the elbows, and in the arm-pits. At this stage a distinctly yellowish staining or discoloration of the general cutaneous surface is observable, like that seen after the fading of ordinary erythematous eruptions. The desquamation begins early in severe cases, being sometimes observed before the sixth day; usually it commences as a furfuraceous or branny scaling about the neck and front of the chest, where the eruption has first appeared. As a rule, it is in proportion to the intensity of the rash, flakes of considerable size being shed from the trunk. The epithelium is shed from the hands and feet in continuous plaques; often a large piece resembling the finger of a glove separates from each digit. Even the nails are sometimes (but very rarely) shed, and the hair falls, and, as in typhus and other severe short fevers, the nails show marked transverse atrophic furrows.

The duration of the peeling stage varies considerably; in patients with thick skins the epithelium, especially of the hands and feet, may not be completely shed till after the expiration of eight or ten weeks. In very mild cases no desquamation may be observed, save upon the hands and feet after several weeks have elapsed. Clement Dukes draws attention to the importance of a careful examination in doubtful cases of the skin over the tips of the ears and the prelabium of the lips, which seldom fails to show evidence of desquamation.

During the stage of eruption the constitutional symptoms increase in severity; the temperature continues to rise usually till the rash is well out, after which it begins to gradually decline. In a typically smart case, upon the fourth day the thermometer registers about 104° in the evening and 102.5° in the morning. In mild cases the temperature rarely rises above 100° . When the throat-symptoms are severe there is no marked evening drop even up to the tenth day, though occasionally the morning temperature may approach to the normal, or be even for a few hours sub-normal. Very severe cases of uncomplicated scarlatina may exhibit a very

high range, and when complications supervene hyperpyrexia may occur. The writer has once witnessed a temperature of 110° before death. Suppurative lesions may prolong the pyrexial period into the third week, whilst as a rule in smart cases without complications it is unusual to find the fever lasting beyond the seventh day. During the pyrexia, all through the eruptive period the pulse-range is very high, the pulse being always quickened out of proportion to the temperature; in cases which recover it may reach 160. In the fatal types of the disease the pulse may register 200 before it becomes uncountable.

The condition of the tongue is usually highly characteristic in all cases save the mildest. The white fur upon the organ in the stage of invasion soon shows numerous red papillæ projecting through the creamy-white epithelium. By the time the eruption has reached its height, the fur has been shed, the process of desquamation commencing at the edges and tip. The appearance is at this stage quite distinctive of the disease: the organ is somewhat swollen, red, raw, beefy-looking, and exhibits the enlarged papillæ giving to the condition the name of the strawberry-tongue, which is well marked by the close of the fourth day.

The throat-appearances, though highly characteristic, vary with the stage of the malady. In the early period of the invasion the mucous membrane is uniformly somewhat reddened and dry, showing slight swelling; but within twelve hours, and often before any eruption is visible upon the neck, a bright, almost erysipelatous, redness is observed all over the area visible to the eye; there is considerable swelling of the membrane, especially over the tonsils and soft palate; the uvula is sometimes œdematous and velvety. The colour deepens as the cutaneous rash develops, and as mucous exudation gathers over the entire pharyngeal surface the swollen tonsils showing white specks over the mouths of the follicles resemble the so-called ulcers which are often mistaken for diphtheritic membrane. But these specks or spots remain discrete, or at most appear to be joined together by a semi-transparent pultaceous exudation of mucus and epithelial debris. The throat-signs usually clear up with the fading of the eruption, but in the anginose type of the disease the tonsils remain livid or purple and swollen, sometimes showing multiple abscesses, and block the entire pharyngeal space. The glands swell considerably, both the salivary and lymphatic, and the cervical connective tissue may in this variety of the disease become the seat of deep brawny swelling or dense board-like induration which in severe cases may break down and slough extensively. These changes occur probably from secondary streptococcal infection, and are accompanied by a high and markedly irregular type of fever. The secretions

of the throat become sanious and offensive, a thin purulent ichor exudes from the tonsils, pharyngeal, and nasal mucous membrane. The intense inflammation may spread to the membrane lining the cavities of the nose and face and, extending up the Eustachian tubes, may set up grave ear-mischief. Perforation of the soft palate is not rare, and when present it always occurs in the pillars of the fauces; but in some cases the entire velum may slough and disappear, the internal carotid artery may become eroded, or a large cervical vein be opened, causing a fatal hæmorrhage.

The usual symptoms which accompany severe fever are always present in smart cases; thus the respirations are greatly increased; headache, restlessness, and mild delirium are common. The urine is scanty and concentrated, the chlorides are diminished, and a small amount of albumin is very commonly present when the rash and fever are at their highest. Albuminuria occurring at a later stage has, however, a different significance, and usually indicates the advent of renal complications or sequelæ. A moderate but marked degree of leucocytosis is usually present. In very severe forms of the disease the constitutional symptoms show marked implication of the nerve-centres; severe delirium, tremors, convulsions, and coma are present, being the result of the action of the toxin upon the brain-substance. In the malignant type of the disease the picture is one of an acute and profound toxæmia, passing into coma and death sometimes before the period of eruption.

TYPES OF SCARLATINA

Scarlatina simplex is the commonest variety of the disease. It is the type included in the description in the preceding pages of the affection when it occurs with slight sore-throat, bright rash, moderate fever extending over a period of about eight days.

Scarlatina anginosa is the term applied to the disease when it appears in severe form with the swelling, lividity, ulceration, suppuration, and sometimes gangrene of the pharyngeal tissue and deep cellulitis of the neck, already described. In this variety of scarlatina the clinical picture presents the obvious symptoms of the streptococcal or other secondary infections grafted upon the scarlatinal toxæmia from the first appearance of the disease. The pulse is very frequent, sometimes registering 160-180 in grave cases; the range of temperature is very high and markedly irregular, showing variations often of 5° to 7° or 8° F. The rash is lacking in brightness, sometimes assuming a livid violet or purple hue, and petechiæ or true hæmorrhagic points are studded through it. When the rash in anginose scarlatina is markedly hæmorrhagic in character, bleeding may occur

from any of the mucous surfaces as in purpura, and the disease is sometimes described under the title of *Hæmorrhagic Scarlatina*; the symptoms rapidly become grave, a typhoid state supervening; such a condition is, however, to be regarded as a variety of the next type of the disease. In anginose scarlatina, recovery generally occurs, but the pyrexial period extends over two or three weeks, and may be even prolonged beyond this by deep cellulitis of the neck, eventuating in abscesses and gangrene. The disease is usually seen in its typical state in children under seven years.

Malignant scarlatina, in its typical condition, is always fatal. The disease is ushered in with rigors, tonic spasms or convulsions, delirium, severe vomiting, and diarrhœa. The temperature may rapidly become hyperpyrexial; ataxic symptoms soon follow, as subsultus, floccitatio, and coma-vigil indicative of the most profound nervous prostration, and the patient may succumb within twenty-four hours after the first symptoms of the invasion have appeared, and before the appearance of the eruption. The entire clinical picture is one of profound toxæmia; in rare cases the nerve-centres may be so gravely deranged that a collapse-temperature maintains till death. If the vital resistance of the patient enables him to struggle against the action of the toxins for another twenty-four hours, an ill-developed livid rash on the body and throat membrane may appear. The victim is stricken down so suddenly in this type of scarlatina that the diagnosis usually can only be made in the presence of an epidemic of the disease or by a knowledge of a family-idiosyncrasy showing marked susceptibility to the virus. It must always be remembered that exposure to the infection from the mildest form of scarlatina may give rise to the malignant type of the disease; and the converse is equally true—an individual may receive the disease in its most insignificant form after exposure to the most virulent malignant type of the affection.

Abortive Scarlatina.—Under this title varieties of the disease are described in which, during an epidemic, the only symptom complained of by the patient may be a slight difficulty or pain in swallowing. The throat is found upon inspection to have its lining membrane reddened and somewhat swollen, and if the thermometer be employed a slight elevation of temperature only may be detected. Such cases are frequently styled *Scarlatina sine eruptione* or *Scarlatina faucium*. Their true nature is sometimes demonstrated by a subsequent typical desquamation, or by the advent of a typical sequela, or by their infecting a healthy patient with the ordinary severe form of the disease. In another class of the abortive type there are no throat-symptoms whatever, only a mild eruption drawing attention to the patient's condition, and there is a very slight pyrexia usually. This variety is known by such names as

latent or *defaced scarlatina* or as *scarlatina fruste* or *scarlatina sine angina*; it may be followed by extensive desquamation, or complicated with rheumatic joint-trouble, or acute glomerulitis.

Surgical scarlatina is the variety of the disease which supervenes in hospital-patients who are suffering from wounds or injuries. It in no way differs from the typical form of the disease save in its extreme mildness, which Caiger attributes to the patient responding to a dose of the contagion which would be otherwise inoperative because of his increased susceptibility owing to injury.

Puerperal scarlatina will be referred to under Prognosis.

Complications and Sequelæ.—*Arthritis* is a common complication; the joints show the usual signs regarded as typical of acute rheumatism. Pain, heat, redness, and swelling attack several joints, especially the smaller ones, usually commencing in the wrists, fingers, or toes, about the period of the fading of the eruption. This is the condition recognised as scarlatinal rheumatism, and there can be little doubt about its close relationship with rheumatic fever, since it is often accompanied by the characteristic complications or other features of that disease, as endocarditis, serous inflammations, subcutaneous nodules, and even *chorea*. It is more prone to occur in adults than in children and in severe than in mild attacks, and is especially liable to appear in rheumatic subjects. Scarlatinal rheumatism may be therefore accepted as due to a multiple infection in which the *diplococcus rheumaticus* plays its part.

This condition must not be confounded with the rarer and grave form of arthritis, also occurring as a complication, which tends towards suppuration of the joints. This suppurative arthritis is a form of pyæmia produced by the streptococcic infection which is so commonly present, as demonstrated in the bacteriological examination of the other suppurative lesions.

Nephritis.—This is the most treacherous of the complications of scarlatina, since it is quite as liable to occur in the mildest as in the most severe cases. Albumin is found in the urine under three different conditions in scarlatina, and these should be differentiated. Thus, when the rash and fever are at their height, a faint cloud may be observed upon the skilful addition of a little urine to cold nitric acid in the bottom of a test-tube; this trace is quite transitory in its duration, and it may not be detected in the next sample. It has no significance as regards the state of the kidneys, and corresponds with the evanescent albuminurias which are observed in all smart febrile conditions. In a smaller percentage of cases, but still possibly in more than one-tenth of all, about *the middle of the third week* albumin is found in small, but in appreciable

amount, and a few tube-casts may be present. Many of these cases leave the physician in doubt as regards the question of their being true examples of nephritis in the strict sense, or of a simple albuminuria. Doubtless the majority clear up completely without hæmaturia, anasarca, or other evidence of nephritis developing. It is wise to regard all such instances of simple albuminuria, potentially at all events, as mild cases of nephritis. In a third form of albuminuria, also occurring during or towards the end of the third week, the symptoms of renal inflammation set in abruptly with all the features of a typical attack of acute Bright's disease. There may be a chill or rigor, severe headache, some pyrexia, and vomiting, with frequent micturition of small amounts of bloody and highly albuminous urine of high s.g., showing many blood and epithelial casts and renal *débris*. Suppression of urine soon occurs in the most serious cases, and uræmic symptoms as twitching, drowsiness, and convulsions, supervene. Occasionally a fulminating type of this complication may be witnessed, in convalescing scarlatina patients who show no symptoms of illness till a fit of uræmic convulsions suddenly supervenes upon a suppressed state of the urine which had escaped even the patient's notice. In these cases there may be no traces of anasarca observed when the convulsion occurs. Usually, however, some œdema appears about the eyelids, hands, or ankles soon after, if not before, suppression has been observed. The tension is high, the pulse quick, and the skin dry and hot.

Between these second and third types of scarlatinal albuminuria an intermediate variety is common in which the symptoms of a mild or sub-acute attack of Bright's disease occur less suddenly; the urine is smoky, of normal or slightly reduced s.g., there is a moderate amount of albumin with epithelial casts, and a considerable degree of general anasarca without any uræmic symptoms. All degrees of scarlatinal nephritis may clear off completely. In a small percentage of cases, however, the disease runs into the typically chronic form, and remains as a sequela till death occurs some years afterwards. When serous inflammation exists with the acute nephritis, the s.g. of the scanty urine may register a higher figure than is ever seen in diabetes.

Pleuritis may occur alone or in combination with other serous inflammations, especially with *pericarditis*; peritonitis is rare; but meningeal trouble, independent of otitis, is not uncommon. Scarlatinal pleuritis is very liable to terminate in *empyema*, which may, moreover, have an insidious onset.

Otitis is perhaps the most frequent of the scarlatina complications. It is very liable to occur in all severe cases in children under seven years. The affection arises as an extension of the throat-inflammation, creeping along the

Eustachian tubes, and before the expiration of the first week causing a purulent otitis media, which, if not promptly incised, leads to perforation or destruction of the tympanic membrane, sometimes ending in total deafness from implication of the labyrinth.

Mastoid abscess may accompany or follow the otitis media, and *meningitis*, *cerebral abscesses*, and *sinus thrombosis* may supervene. *Panophthalmitis*, *albuminuric retinitis*, and sometimes *diphtheritic conjunctivitis* occur.

Heart.—The complications affecting this organ are more liable to occur when rheumatism or arthritic trouble is also present. Endocarditis and pericarditis both occur; but, as a rule, the degenerative changes in the cardiac muscle are less obvious than in acute rheumatism, save in those cases where the scarlatinal toxin causes hyperpyrexia.

Lung.—Bronchopneumonia and bronchitis are by no means so common as in measles; in the acute anasarca the lungs or the larynx may become oedematous.

Adenitis.—The swelling of the cervical lymphatic glands already mentioned is a feature of the anginose form of scarlatina, and cannot be regarded as a complication; indeed, the same may be said of the diffuse brawny cellulitis, parotitis, gangrene, and abscesses which form. In some cases of simple scarlatina, however, all the lymphatic glands in the cervical region and about the angles of the jaws towards the end of the attack become enlarged and indurated, in which condition they may remain for many months before showing signs of resolution or suppuration.

Diphtheria is a serious and by no means uncommon complication or coincident affection. It may appear almost along with the scarlatinal eruption; the writer has witnessed in several cases a genuine diphtheritic membranous exudation on the fauces and tonsils before the appearance of the characteristic rash of scarlet fever. Commonly, however, diphtheria, when it complicates scarlatina, appears during the later stage of desquamation of the skin of the trunk; hence its common title 'post-scarlatinal diphtheria.' The frequent coincidence of measles and scarlatina, as reported in hospital-statistics, is explicable, according to Dukes, on the theory of confusion in the diagnosis. He points out that *rötheln* and the 'fourth disease' are frequently concurrent or consecutive.

Nervous System.—The chief symptoms caused by the action of the toxins on the nervous system have been already mentioned, as convulsions, coma, &c., but phenomena referable to the cerebrum and spine are sometimes met with, which can more correctly be considered as complications. Of these *chorea*, *sinus thrombosis*, *meningitis*, *cerebral abscess*, *infantile hemiplegia*, and *atrophic spinal paralysis*, resembling

the poliomyelitis anterior acuta type, are the most noteworthy.

Ulcerative or gangrenous *stomatitis* is sometimes observed, and the gangrenous or sloughing process may attack the female genitals.

Diagnosis.—In a typical case the diagnosis cannot easily be mistaken. It is in the mildest forms of the disease, and in the malignant type, that a difficulty usually arises. In the latter, as already stated, death often occurs from profound toxæmia before the rash has time to develop, and even the throat-manifestations may be absent or but imperfectly marked, in which case the diagnosis must mainly rest upon the history of a recent exposure and of the family-idiosyncrasy to the disease.

In the mild forms, the diagnosis may be impossible till after the fourth day, when the epithelial fur of the tongue strips off, and a little later the skin begins to peel from the tips of the ears, lips, and face. It is therefore imperative that in all doubtful cases the most rigid isolation should be observed till after the end of a week at least.

Measles is differentiated by the longer duration of the stage of invasion, the eruption not appearing in that disease till the evening of the fourth day ; during this period the characteristic catarrhal and coryzal symptoms are obvious, and Filatow's or Koplik's spots are visible upon the buccal and palatal mucous surfaces. The eruption first appears as raised rounded papules, which, running together, form patches of crescentic shape, divided by areas of normal skin, markedly contrasting with the confluent punctate eruption of scarlatina. In the rare cases of severe measles, where the eruption is confluent, the face is densely covered and somewhat swollen, and the rash is never of so bright a red or boiled-lobster hue as in scarlatina.

Rötheln, *Rubella*, or *Rose-rash* has usually no prodromal symptoms till the rash appears ; if mild malaise is complained of in a severe case, the eruption will follow in a few hours. This appears as small rose-dots, larger than the scarlatina puncta, first coming out behind the ears and around the mouth and on the scalp, whence they spread over the whole body ; the rose-specks coalesce into spots or small patches, but seldom become so diffuse as in scarlatina, in which respect they more closely simulate a mild measles-rash, but they are not crescentic. The patient usually has no sensation of illness, even when the rash is extensive and well-developed. The most characteristic diagnostic feature is the presence of a universal enlargement of the lymphatic glands throughout the body ; these may be felt not only in the neck, but in the arm-pits and groins, to be slightly tender and indurated. Though the mucous membrane of the throat is somewhat

reddened, there is no secretion or marked swelling with ulcerous spots on the tonsils, as seen in scarlatina. Desquamation never occurs except in rare cases as a slight furfureous powderiness, and there are no sequelæ or complications.

The '*Fourth Disease*' of Clement Dukes more closely simulates mild scarlatina. This observer insists upon the affection being a separate entity, differing essentially from rōtheln, measles, and scarlatina; hence its provisional title. The disease is characterised by a scarlatiniform, rosy, diffuse eruption, spreading over the entire body in a few hours, preceded and accompanied usually by no symptoms of illness, though the throat is velvety and reddened, and the lymphatic glands throughout the body are enlarged and tender, and the conjunctivæ are pink. The essential diagnostic feature from the last-mentioned disease (rōtheln) is in the desquamation which usually follows; this is often as complete and extensive as in scarlatina. The peeling, however, bears no proportion to the severity of the eruption, and seldom occurs in sheets. From the rash of scarlatina, Dukes points out that it differs in its bright rosy-red colour and in the fact that the skin never presents to the touch the pungent burning sensation conveyed by scarlatina. The raspberry or strawberry tongue of scarlatina is never associated with the disease, which affords no protection or immunity from scarlatina, rose-rash, or measles; it often occurs in scarlatina epidemics, however, and attacks a patient convalescent from that disease, whilst in other cases the scarlatina follows an attack of the '*fourth disease*.'

Acute tonsillitis, or *quinsy*, is differentiated by the following points: Vomiting rarely, if ever, ushers in an attack; the affection is unilateral at first; the second tonsil, when involved, shows its swelling at a later stage or after an interval; should, however, both be swollen, one is certain to be more acutely inflamed than the other. An eruption sometimes accompanies tonsillitis, but when this is the case it is always of the erythematous type, and never consists of papules or punctate dots. Though the tongue is furred, peeling of its epithelium never occurs upon the fourth day, so that the characteristic strawberry-tongue is absent throughout; there are no sequelæ, and desquamation does not occur.

Diphtheria, as already stated, sometimes co-exists with scarlatina, in which case symptoms and signs of both diseases will be present. Scarlatina cannot, however, be mistaken for diphtheria, unless when the exudation is very extensive and covers the tonsils with a thick pultaceous coating. This, however, never peels off like the diphtheritic membrane, and upon examination does not exhibit the Klebs-Loeffler bacillus. Diphtheria is sometimes accompanied by an eruption, in which case it is very liable to be mistaken for

scarlatina, but only if seen after the eruption has faded. The rash, when present in diphtheria, is always erythematous, never punctate; it is very transient, and appears only upon the chest or abdomen or neck.

Small-pox eruption can never be mistaken for scarlatina; but in the early stage of variola, with the high fever, an erythematous rash is often observed over the lower part of the abdomen, arm-pits, and inguinal region. This prodromal rash may be accompanied by some redness of the throat. The diagnosis will be cleared up in twenty-four hours by the appearance of the shotty papules; but before then the severe pain in the back which is so characteristic of the disease should settle the diagnosis in the presence of an epidemic.

Acute rheumatism may be diagnosed when the arthritis of scarlatina is unusually severe and the eruption has faded; but ordinary care will prevent such a mistake when the throat and characteristic strawberry-tongue are noted; a search should be made for desquamation commencing about the ears and lips. Scarlatinal rheumatism is, moreover, never accompanied by the characteristic profuse sour-smelling perspiration observed in typical uncomplicated rheumatic fever.

Pityriasis rubra may be suspected if the case is first seen in the freely desquamating stage, and even the early stage of scarlatina may be closely simulated by an acute attack of this form of dermatitis, when it begins with a severe rigor, high temperature, and the appearance of a diffused red rash rapidly covering the entire cutaneous surface. There are no red puncta in this rash, and the throat and the tongue signs of scarlatina are absent. In scarlatina one feature of the desquamation usually is present, and proves a valuable aid in diagnosis in many cases. This is the appearance of minute white points in the horny layer of the epithelium, which, on being shed, become pin-holes. Each point corresponds to the position of a swollen papilla, and the surrounding circles of epidermis coalesce as the process extends, so that large irregular-shaped patches are thrown off. In the two types of acute exfoliative dermatitis—the large and the small scale desquamation—the thin papery epidermal scales are formed again and again, the desquamating process being continuous, so that such a case cannot be confounded with one of scarlatina in the peeling stage, if the skin be watched for twenty-four hours or more. In this form of dermatitis the nails are often shed, which is an extremely rare event in scarlatina.

Prognosis.—The mortality of scarlatina varies widely in different epidemics. There cannot be any doubt that during the last third of a century or more the type of the disease, as usually met with, is becoming progressively milder. The general mortality for all ages, as seen in the records of fever-hospitals, gives an erroneous idea of the death-rate in the

disease, since the mildest cases figure but slightly in such statistics. Probably the general mortality of private and hospital cases taken together would fall well under 2 per cent. As already incidentally mentioned, the mortality is strikingly influenced by age. In infants under one year, the prognosis is grave, and it should always be regarded as serious, except in the mildest type, up to the third year of life. After the fifth year it markedly falls. Family-idiosyncrasy, when known to exist, deepens the gravity of the prognosis, even when the child has been protected from exposure up to the age of puberty.

In forming a probable forecast the rule may be relied upon generally, that the severity of the attack is in the main proportionate to the intensity of the eruption; but the super-vention of nephritis, otitis, rhinorrhœa, heart-complications, and cellular abscesses often occurs at a later stage in the mildest forms of the disease, so that the prognosis in scarlatina should be always guarded. A livid eruption, following an onset in which the vomiting is incessant and the pulse very rapid, is a grave sign. The anginose type must always be regarded as serious, and especially so if the eruption shows hæmorrhagic or petechial characters, and the pyrexia be prolonged into the middle of the second week. Delirium in adults is a serious symptom, and often is indicative of the super-vention of a secondary streptococcal infection. For the same reason a septic rash appearing after the fading of the primary eruption must always be regarded as a very grave omen in patients at any age.

Puerperal scarlatina is the name given to the disease when it occurs immediately before or soon after delivery. It is of a very fatal type, generally terminating in death from septicæmia, even when the most rigid antiseptic precautions are maintained from the onset of the invasion. When the disease attacks pregnant women before the advent of the eighth month, labour seldom is precipitated, and such cases usually pursue a satisfactory course, but the danger enormously increases as the onset is delayed towards the end of the natural term of gestation. It is proportionally intensified by the shortness of the period elapsing between delivery and the onset of an attack of the disease.

Malignant scarlatina always ends in a fatal issue, usually before the end of the third or fourth day. More than half the cases of post-scarlatinal diphtheria died in the days before the serum-treatment; the mortality has been markedly diminished by early resort to injections.

Treatment.—*Preventive* or prophylactic measures, as mentioned under the Etiology of the disease, are of more importance in scarlatina than in any other infectious fever, since the mortality of the disease is so obviously diminished by the

age of the patient. " Rigid isolation should be insisted upon from the first hour of the invasion. The disease is, in the writer's opinion, contagious at this early period ; but Clement Dukes states that of all the zymotic diseases scarlatina is the least infectious in its early stage. Unquestionably the period of most intense contagiousness corresponds with that of the full development of the rash, and not with the stage of desquamation, as formerly held, and still adhered to by a few authorities. Nevertheless, the rule should be rigidly enforced of at least a six weeks' quarantine in all cases. The discharge from the nose of a purulent rhinorrhœa should prolong this period considerably. Much has been written about the significance of so-called 'return' cases. By this term is meant the cases of scarlatina which occur in a household after the convalescent patient has been sent back to his home, or the cases which arise amongst the returned children who have been temporarily banished from the home in which the scarlatina-patient has been treated. The evidence of such late infectiousness must not be accepted as proof of the virulence of the remnants of the desquamating epidermis, since it is proved that fomites attaching themselves to clothing, furniture, &c., may retain their activity for very long periods.

By the practice of early inunctions of eucalyptus oil or other harmless antiseptics, in conjunction with repeated baths containing a sufficient amount of permanganate of potassium to distinctly tinge the colour of the skin, the danger of infection becomes practically *nil* after at latest a period of three weeks ; though, to make assurance doubly sure, it is wise to insist upon the six weeks' isolation period.

It is needless to dwell upon the obvious importance of the most rigid disinfection of all clothes, bedding, and every utensil used in the sick-room ; of the disinfection of all the excreta of the patient, and of the clothing and persons of nurses and attendants coming into contact with the disease. Many cases occur from fomites adhering to wall-paper or lying in the joints of the flooring of the sick-room, and it is not sufficient to fumigate with sulphur, carbolic acid, or formalin—the walls should be stripped, re-papered, or painted, and the woodwork carefully disinfected and covered with fresh pigment.

There is no known drug which exercises a destructive influence over the scarlatinal virus once the latter has obtained an entrance into the body ; the treatment of the disease therefore till the discovery of an efficient anti-scarlatinal serum must remain entirely symptomatic. In the author's 'Dictionary of Treatment' (4th ed.), pp. 865 to 870, will be found details concerning the management of the patient as regards diet, sick-room arrangements, disinfection, in-

unctions, &c. Only general principles need be here referred to. Absolute rest in bed for a period of at least three weeks is advisable in even the mildest cases in order to diminish the risk of complications. The diet should be mainly milk, light farinaceous foods, and weak soups. For routine administration a simple diaphoretic mixture containing *Mindererus* spirit or sweet nitre is unobjectionable, and tends to minimise the burning dryness of the skin. As soon as the eruption has commenced to fade the entire surface of the body should be rubbed lightly with an anointing oil (carbolic acid 1, olive oil 50, or preferably a 1 in 5 eucalyptus oil). It is a wise precaution to attend to the condition of the mouth, throat, and nose even in the mildest cases, in order to minimise or prevent septic complications. This is best carried out by the use of a 1 in 100 carbolic spray in rose water.

Complications as they arise must be treated on recognised therapeutic principles: high fever or hyperpyrexia by judicious tepid sponging or cold packs, nephritis by hot packs, hot-air baths, or other diaphoretic agents; cardiac failure by stimulants, &c. Otitis demands prompt treatment, suppuration being relieved by a resort to early incision of the tympanum and a subsequent scrupulous use of mild antiseptic solutions injected with gentleness and skill; abscesses under the dura mater and mastoid abscesses should be opened freely and early by a deep incision or by the more radical operation first carried out in 1883 by Dr. Joseph Nelson, who gouged out the mastoid successfully and entered the cranial cavity, evacuating a quantity of pus under the dura mater after the patient had been comatose for some time, though there was no evidence of external suppuration in the case.

Abscesses in the tonsil or deep cellular tissue of the neck demand equally prompt and thorough evacuation.

SCIATICA

This is the term applied to several different affections the main or only symptom of which is severe pain suffered in the region of the sciatic nerve.

Etiology.—The symptom-complex recognised clinically by the above somewhat indefinite term, whatever may be the pathological condition underlying it, commonly follows an exposure to cold and damp. Men are much more frequently affected than women, and the usual age is somewhere between forty and fifty. The exposure preceding the attack is often found to be such as a prolonged sitting posture on cold damp grass or upon a vehicle when the garments are saturated with rain. The attack is much more liable to occur under these conditions if they have followed upon prolonged muscular

exertion, as in walking, climbing, rowing, &c., and if the seat upon which the patient is resting be such that its edge will press directly upon the nerve, and if the individual has permitted his damp garments to dry upon his body. Sometimes the attack has been known to follow a severe contusion on the buttock.

All authorities agree that rheumatism and gout are pre-disposing causes. Pelvic diseases of whatever kind which produce pressure upon the nerve-trunk inside the body or at its exit through the bony notch are responsible for a considerable percentage of cases.

Pathology.—(1) By most authorities sciatica is regarded as a *neuritis* of the sciatic nerve, corresponding in the main to the inflammation met with in other nerve-trunks, and described under 'Neuritis.' That this is true of the great bulk of all cases commonly met with is probable from the frequency with which the attack has suddenly followed exposure, which is regarded as a common cause of other recognised forms of mono-neuritis, and also by the fact that portions of the nerve excised for the cure of the affection are usually found to be in a state indicative of interstitial neuritis. Closely allied to the changes observed in this interstitial inflammation are those found in other cases where the fibrous tissue surrounding the nerve is in a state of acute fibrositis often leading to adhesions between the nerve-sheath and the surrounding connective tissue of fascia, muscles, &c. This is, in the writer's opinion, the pathogeny of those by no means uncommon cases where the sciatica is associated with a severe attack of lumbago or of so-called muscular rheumatism affecting some other region rich in white fibrous tissue.

(2) Another view still held by many, and formerly accepted with little reservation, is that which regards sciatica as a true *neuralgia* of the nerve—a purely functional affection identical pathologically with the functional neurosis which is so commonly seen affecting the fifth nerve. Though a true neuralgia of the sciatic nerve or of its component cords does exist, and may be differentiated clinically from the neuritic cases, there can be little doubt that few cases of the disease are of this nature; the majority exhibit the usual clinical features of an inflammatory state of the nerve.

(3) The view is maintained that the majority of the cases are due to pressure within the pelvis or about the notch, which may induce either a secondary neuralgia or a secondary neuritis of the trunk of the sciatic. Uterine, ovarian, rectal, or other tumours, a loaded rectum, or the pressure of the foetal head during a protracted labour, may undoubtedly cause sciatica. Osler believes that the affection is often produced by chronic arthritis of the spinal column.

(4) Nothing better illustrates the difficulty in arriving at a satisfactory working pathological hypothesis than the view recently put forward by Dr. W. Bruce, who, after a study of 418 examples of the disease, maintains that the nerve is not the seat of the mischief, as the trouble is always located in the hip-joint, whose capsule was tender to pressure in more than half the cases. This view is supported by the well-recognised fact that marked wasting of the glutei, with more or less obliteration of the gluteal fold, is frequently observed in sciatica; and Bruce points out that if the sciatic nerve itself was affected, *all* the muscles supplied by it should be wasted, instead of the glutei only, which receive their supply separately from nerves arising from the sacral plexus. The pain along the outer side of the leg, reaching sometimes to the heel, and the accompanying patches of anæsthesia, he maintains are 'referred' phenomena due to the implication of the sciatic branch which supplies the hip-joint.

Symptoms.—Pain constitutes the chief symptom of the affection; it sometimes comes on with marked abruptness, though a much less severe and sudden onset is the rule. Its distribution corresponds in the main with that of the sciatic nerve, which, commencing as a prolongation downwards of the chief part of the sacral plexus, leaves the pelvis through the great sacro-sciatic foramen, courses down the back of the thigh, to divide into its two chief branches—the external and internal popliteal.

The character of the pain is described in terms as various as are the temperaments of the victims of sciatica. It is aggravated by movements of all kinds; sometimes spontaneous pain is entirely wanting, but often this is counterbalanced by an extreme degree of local fidgetiness which compels the patient to shift the position of the affected limb, bringing on an acute paroxysm of severe suffering. In other cases a dull constant aching is experienced in the trunk or branches of the nerve, which the patient describes as gnawing, boring, or burning. In mild cases, where locomotion is not prevented, both these kinds of pain are commonly present; the patient walks lame, suffering considerably during the attempts at locomotion, and for many hours, or even days, afterwards the continuous pain during complete rest remains along the back of the thigh, over the calf, and along the outer side of the leg to the heel or sole. Walking can often only be accomplished with the knee somewhat flexed, the toes only reaching the ground. In the very acute cases with abrupt onset a characteristic symptom, which is usually agonising in its intensity, is witnessed: as the patient drops over asleep, a violent spasm of the muscles of the limb occurs, accompanied by an electric-shock sensation down the nerve-trunk and its chief branches which compels the patient to scream.

The pain is commonly most intense in the following localities—viz. above the hip in the region of the sacro-iliac articulation, about the notch, in the popliteal space, below the head of the fibula, over the external lateral ligament of the ankle-joint, and on the outer part of the dorsum of the foot. Tenderness in the nerve or in any of its branches may be often elicited by even moderate pressure.

In sciatica depending upon a pure *neuralgia* of the nerve, 'tender points' may be detected when the trunk of the nerve itself is quite free from pain on pressure. These spots correspond with those regions in which the patient experiences the most acute suffering—viz. the posterior iliac spine, the notch, below the head of the fibula, and a spot on the dorsum of the foot. The pain in sciatic neuralgia usually tends towards the type of intermittency or even periodicity, and it is not unusual to obtain evidence of true neuralgia or a distinct history of such in some other nerve. There are no areas of anæsthesia, and wasting of the muscles, if present, is only such as may be accounted for by the temporary disuse of the limb. Purves Stewart points out that the cremasteric reflex may be exaggerated upon the affected side, but the deep reflexes are unaffected.

In *neuritis* or perineuritis of the sciatic nerve there is tenderness along the trunk or branch affected; the pain, though liable to acute exacerbations on muscular movement, is of the continuous type. There are other evidences of severe nerve-implication, as tingling, numbness, and formication; areas of more or less complete anæsthesia may be detected in the skin over the back of the thigh or outer side of the leg and foot. Certain groups of muscles, as the hamstrings, gluteus maximus, calf, or peronei, exhibit marked wasting and sometimes fibrillary twitchings. The ankle-jerk is absent or diminished on the affected side, but only in the most severe and protracted cases is the reaction of degeneration to be detected. A certain degree of real weakness or loss of power in the muscles is always present in cases of long standing, and, as insisted upon by Anstie, this is not merely the result of disuse. When the neuritis is confined to the small sciatic, which is rarely the case (as this branch when affected usually suffers with the trunk), the anæsthetic areas are confined to the skin over the buttock or upper part of the back of the thigh, and there is no wasting below the knee.

The Course and Duration of the affection will be referred to under Prognosis.

Diagnosis.—The typical *neuralgic* symptoms, when present, leave little doubt about the diagnosis, but care nevertheless is requisite in excluding some reflex source of the neurosis. Thus, as a true facial neuralgia may be caused by the presence of a carious tooth which may itself be painless, so a latent

hip-joint or sacro-iliac mischief might be the exciting cause of a true neuralgic sciatica.

Neuritis of the nerve is recognised by the features already described; the general tenderness of the nerve-trunk and the exquisite pain caused by passive flexion of the limb which puts the nerve on stretch are highly characteristic. The main point in the diagnosis of these cases is to determine whether the neuritis is primary or secondary. The primary nature of the affection can only be accepted when pelvic disease, tumours, joint-mischief, &c., can be eliminated. The writer has elsewhere pointed out that, owing to the shape of the modern water-closet seat, an inflammation of the bursæ about the sacro-sciatic notch is sometimes produced, especially in subjects of constipated habit, and this *bursitis* is apt to be mistaken for sciatica. It is differentiated by the absence of the usual evidence of tender points in the skin or tenderness along the course of the nerve, and the pain is elicited by bringing into action the muscles whose tendinous extremities pass out through the notch.

Chronic rheumatism closely simulates sciatica in many cases, and, indeed, often a differential diagnosis is as impossible as it is unnecessary, since the fibrositis, when it invades the fasciæ, muscular sheaths, and aponeuroses of a lower extremity, is very unlikely to entirely spare the white fibrous tissue in the sheath of the great sciatic nerve. The discovery of the indurations, cords, or nodules found along the course of the intermuscular septa or other fibrous structures is nevertheless of great importance, since it furnishes a clue to the pathology of the accompanying sciatica. For the same reason the recognition of any degree of associated lumbago is very important.

Sacro-iliac-joint disease may cause radiating pain which is sometimes mistaken for sciatica, but the localised tenderness and swelling over the synchondrosis to be detected by careful palpation is very characteristic, as is also the fact that pain can be produced at the joint by strong pressure made upon the anterior-superior spine or iliac crest.

Hip-joint disease is eliminated when pressure over the great trochanter and force applied to the femur so as to press the head of that bone against the acetabulum cause no pain. *Thrombosis of the femoral vein*, associated with severe pain over the calf-muscles, may be differentiated by a careful palpation of the vessel below Poupart's ligament. Affections of the *cauda equina* cause pain and sensory disturbance in both lower limbs, whilst sciatica is always unilateral; but it is worth remembering that occasionally, as pointed out by Fagge, when a paroxysm of sciatic pain is at its height the pain may be reflected along the sciatic nerve on the sound side.

Prognosis.—This is often most difficult as regards the question of duration; the general tendency of the disease is towards complete recovery, but the attack may last for weeks, months, or even years. The severity of the initial symptoms are often out of proportion to the duration of the affection; a very formidable attack sometimes ends in complete recovery within a few weeks, whilst an apparently mild seizure may drag on for many months. Where marked wasting, spasmodic contraction of the muscles, fibrillary twitchings, and any change in the electrical reaction are present, the convalescence is certain to be very protracted and liable eventually to be incomplete. The likelihood of relapses and recurrences must be borne in mind, and the writer has repeatedly observed a remarkable fact in this connection—viz. that a patient who has once suffered from sciatica is very liable to have a minor attack should he go to sleep in his ordinary wearing apparel, even when covered up carefully with wraps or bed-clothing.

Treatment.—In all cases at the beginning, whether they be mild, or severe, absolute rest in bed is essential. This rest may be further ensured by the application of a long splint so as to prevent any muscle supplied by the nerve being brought into action.

In the so-called rheumatic cases, aspirin, salicylates, or any of the newer analgesics are indicated in the acute stage, whilst later on the routine drug should be iodide of sodium in large doses.

The chief indication is found in drugs for the relief of the severe pain; of these there can be no second opinion that morphine in the form of hypodermic injection is the most reliable; though, of course, the danger of the opium-habit is never to be lost sight of. *Acupuncture*, carried out by thrusting a needle deeply through the skin so as to penetrate the nerve in several places; *aquapuncture*, or the deep parenchymatous injection of sterilised distilled water; and *counter-irritation* over the course of the nerve by a series of small blisters, are all beneficial. The writer's method of treating the disease consists in combining in one manœuvre all these agents in the following manner: A full hypodermic dose ($\frac{1}{4}$ to $\frac{1}{2}$ gr.) of morphia hydrochloride is dissolved in about half to one drachm of water, and the sterilised solution is put into a large hypodermic syringe, to which is attached a stout, long, hollow needle. The course of the nerve having been mapped out on the thigh and leg, the stopper of a bottle containing pure liquefied carbolic acid is laid upon the skin in half a dozen or more places over the line of the affected nerve. This at first causes local anæsthesia (followed in some hours by vesication), which permits of the painless insertion of the needle thrust rapidly into the tissues at right-

angles to the skin till the operator feels that he has probably punctured the nerve; 5 to 10 mins. of the solution are then injected at each puncture. This method he sometimes combines with yet another plan—viz. *nerve-stretching*, by powerfully flexing the thigh upon the pelvis either before, during, or after the injection-process. Cocaine or β -eucaine may be substituted for or combined with this morphine treatment in special cases where the opium-habit is to be specially guarded against.

In chronic obstinate cases osmic acid may be injected in the same manner, sometimes with most gratifying results; the drug, however, frequently causes very severe pain, so that it is often a good plan to inject it separately into the nerve in a couple of places between the punctures made for the morphine-solution.

Upon pages 592 to 611 in the author's 'Dictionary of Treatment' (4th ed.), the different members of the list of drugs useful in neuralgia and neuritis are discussed, and massage, hydro-pathy, electricity, neurotomy, neurectomy, and other agents are referred to.

SCLEREMA NEONATORUM

This rare affection is characterised by a remarkable induration of the skin, which may be congenital or may appear within a few days after birth, and is often associated with grave constitutional symptoms indicative of alimentary or pulmonary disturbance.

Two different conditions are frequently described under the term 'sclerema,' which, though in very rare instances they may occur together, nevertheless should be regarded as essentially different in many respects. These are Sclerema neonatorum and Œdema neonatorum. The main clinical symptoms common to both affections are a greatly depressed body-temperature and cardiac weakness, associated with the skin-induration.

Sclerema neonatorum proper is described by Langer and Brissard as *Fat sclerema*. The disease shows itself a few days after birth usually in prematurely born weaklings, but evidence of its presence is sometimes observed at birth even in stillborn infants. It first shows itself over the back, buttocks, and lower extremities, from which it spreads so as to involve the subcutaneous tissue of the entire body before the end of a week. The skin at first presents a yellowish-white waxy appearance as if the fat was frozen; it then becomes dusky or livid, and so indurated, tense, cold, and brawny, or hard that it cannot be pinched by the fingers and cannot be made to pit on even prolonged pressure. The rigidity of the body

when the affection becomes general interferes with all movements: the mouth cannot be opened and sucking is impossible, the child invariably dies before the end of a week or ten days, the temperature, pulse, and respirations being all markedly depressed. In a small percentage of cases the induration and adhesion of the skin to the subcutaneous tissues is limited chiefly to the posterior aspects of the body or to the limbs, and the infant may by careful feeding and warmth recover by the end of several months. The disease was more common in the period when defective hygiene, improper feeding, and overcrowding were the usual characteristics of foundling-hospitals.

It has been so frequently associated with bowel-complaints, vomiting, and pulmonary affections which depress the general vitality, that some authorities, including Parrot, have been led to explain the pathogeny of the condition as arising from desiccation of the skin and thickening of its layers, with or without proliferation of the fibrous tissue supporting the fat-cells. Langer, upon the other hand, regards the affection as due to the solidification of the fat, which becomes solid in infants at a lower temperature (*i.e.* about 90° F.) than that of the adult adipose tissue, which does not solidify till reduced to 32° F.

Edema neonatorum, sometimes called *sclerœdema*, appears also in some cases at birth, but more usually attacks prematurely born weak infants whose circulatory apparatus is feeble, probably as the result of malnutrition of the mother. The indurated œdema appears usually within a few days after birth, appearing in the legs, back, hands, and genitals, which become dense, cold, rigid, and livid. The skin, though very hard, pits on firm and prolonged pressure, and it can be pinched up in folds; there may be slight jaundice. The induration tends to confine itself to the most dependent parts, and seldom, if ever, does it become universal, as in true sclerema. With the œdema and lividity there is always a marked depression of the body-temperature and of the pulse and respiration rate, drowsiness supervenes, and the infant usually dies from asthenia; but, more frequently than in sclerema, tedious recovery supervenes under judicious treatment, unless vomiting, diarrhœa, or pulmonary collapse sets in.

The essential difference between the two affections is the presence of a true serous effusion into the cellular tissue in sclerœdema, though in both conditions the adipose tissue is markedly firm or indurated.

The pathogeny of the affection is variously held to be the result of the depressed circulation, lymph-obstruction, microbic toxæmia, pulmonary embarrassment, venous thrombosis, or nephritic mischief. A connection between the two conditions

is seen in the occasional supervention of marked œdema in simple localised sclerema.

Diagnosis.—The only condition liable to be confused with these affections is *sclerodermia*, but this is easily eliminated, since it never occurs in newly born infants; the condition is described in the succeeding article.

Prognosis.—Both conditions are generally fatal in foundling-hospitals, sclerema being invariably so when it appears in the universally distributed form. The outlook is brighter, however, in sclerœdema, some authorities quoting statistics which show a mortality of only 50 per cent. in cases occurring in private practice.

Treatment.—This resolves itself into the prompt establishment of a complete change in the infant's environment. An incubator should be resorted to, increased warmth being essential, and forced feeding being indicated when sucking is impossible. At a later stage gentle massage should be perseveringly tried.

SCLERODERMIA

Also known as Hide-bound Disease, is an affection characterised by a board-like or wooden induration and rigidity of the skin, either of partial or universal distribution.

Diffuse Symmetrical Sclerodermia.—This is a very rare disease. It usually commences without any prodromal symptoms, the first sign of the affection being the induration and stiffness of the skin in the affected areas, but sometimes numbness, tingling, and other sensory disturbances herald in the skin-rigidity. The disease may occur at any age after the infant-period; the majority of the cases are seen in middle-aged women, but, though the affection is styled sclerema aduitorum to distinguish it from the condition described in the previous article, many cases have been reported as occurring in childhood. Of its etiology little is known, save that exposure to cold, prolonged mental worry, Graves' disease, rheumatism, erysipelas, and Raynaud's disease have preceded the onset in many cases. Of these antecedents the last-named is the most important; many observers, including Stephen Mackenzie, Ewart, Wills, and Griffiths, have reported of late years the close association of the two diseases.

Symptoms.—The skin is usually first felt to be stiff and hardened about the face, upper extremities, neck, shoulders, or thorax; seldom are the lower parts of the body involved at the commencement of the disease. More or less symmetrical areas are invaded, and in acute cases the entire body may be affected in a few weeks; more usually the extension is gradual. The skin feels indurated and wooden, it cannot

be pinched up between the fingers; in the early stage of some cases prolonged pressure causes marked pitting, but this sign eventually disappears as the induration increases, and the adhesion of the skin to the underlying structures becomes more evident. To the eye the affected cutaneous regions seem little altered, but some glazing, smoothness, and obliteration of the natural lines may be detected; conglomerations of small dilated vessels, with or without patches of erythematous redness, may be observed in some cases, in others patchy pigmentation as deep as that observed in Addison's disease may exist; but, as a rule, the general hue of the affected surface is pale and waxy-looking. To the touch the skin is dry and cool, and at a later stage becomes rough from shedding of the cuticle, especially on the legs. The borders of the affected areas are not sharply defined from the healthy skin, but may be vaguely mapped out by palpation. Sometimes vaso-motor disturbances are visible, suggestive of the local cyanosis seen in Raynaud's disease, and severe arthritic pains may be complained of at the commencement of the disorder. As a rule, cutaneous sensibility is little interfered with.

The hide-bound condition shows itself in the diminished mobility of the parts covered by the rigid skin; the joints of the limbs may become practically immobile, and as atrophic shrinking of the indurated skin follows at a later stage the limbs may become flexed. Thus the joints of the fingers and hands become permanently deformed and ankylosed, the condition known as *sterodactylia* being produced, the third and fourth fingers are deeply flexed on the palm, whilst the thumb-phalanges are over-extended and the first phalangeal joints of the index and next finger are flexed. The fingers are shortened and wasted and the skin over them indurated, pallid, or sometimes pigmented. The immobility of the chest-wall interferes with respiration, and deglutition is rendered difficult. The most remarkable feature in the disease is the expressionless condition of the face from the rigidity of the skin; the lips are immovable, the mouth cannot be opened, mastication becomes difficult or even impossible, and the eyelids in rare cases remain widely open and immovable.

This statue-like expressionless physiognomy at a later stage may give way to a more serious deformity, in some cases proceeding to an extent which becomes gruesome. As the indurated skin shrinks, its pressure causes wasting of the underlying fat and muscles; the tightened skin then appears adherent to the bones, the lips become shortened, the gums shrivel, causing shedding of the teeth, and the nostrils are compressed. This is the *atrophic* stage, as described by Crocker, who believes that it only supervenes upon the form of scleroderma which begins with firm doughy œdema,

and does not follow in those cases which primarily commence with hard, dense, non-œdematous infiltration. The skin in this phase of the disease is of ivory-whiteness, and owing to its wasted tightened state, the bones, atrophied muscles, and tendons are outlined through it; pigmentation may occur in irregular areas, and ulceration is liable to supervene from accidents to the atrophic cutaneous tissue, and the phalanges may even necrose.

The duration of the disease, according to most observers, varies in the densely indurated and in the œdematous forms of the disease. In the former the tendency is towards slow resolution, occupying months or years, after which the skin becomes normal. In the œdematous cases contraction follows as atrophy progresses, and though recovery may occur after many years of a stationary condition, the wasting of the underlying structures, the contractions, and fibrous ankylosis remain as permanent deformities.

Pathology.—No satisfactory working hypothesis has been yet discovered; in the absence of such, it is held by many that a local blocking of the lymph and blood supply explains a considerable portion of the clinical phenomena. Touchard and Alquier have recently verified the observations of former observers who described a sheath-like layer of elongated connective-tissue cells surrounding and blocking the small vessels. These authorities, in addition, found degenerative changes in the inner lining and in the vascular walls, from which they reason that the point of departure consists in a toxæmia, the poisoned blood setting up in the first place a perivascular sub-inflammatory or irritative action. Other authorities, from the apparent resemblance of the diffused form of the disease to a myxœdematous condition, have suspected the thyroid gland of playing the chief rôle in the production of the sclerosis; but the research of Tedeschi shows that the general metabolism is undisturbed. Crocker regards the sclerosis as the outcome of the endarteritis, which he surmises has its origin in the nervous system in the region of the vaso-motor centre, the anatomical changes being mainly confined to the corium and subjacent tissues, whose connective and elastic tissue elements are increased and the organic muscular fibres hypertrophied.

Diagnosis.—The disease resembles the *sclerema* and *sclerœdema* already described as occurring at birth or immediately afterwards in weak and often prematurely born infants; from these it is readily differentiated by the age of the patient, as true sclerodermia never occurs in infancy. From *myxœdema* it is easily differentiated by the absence of the thyroid signs, of the mental condition, of the state of the hair, and of the supra-clavicular swellings. *Addison's disease* is only likely to be suspected when the pigmentation is extreme;

but the anæmia, characteristic feebleness of the heart, and vomiting are absent.

Prognosis.—In most instances the disease runs an extremely chronic course, sometimes resolving after many years. The prognosis, as already stated, is better in the plump or indurated cases than in the atrophic form, and less favourable, as pointed out by Crocker, in the cases which show marked œdema, since these are liable to eventuate in atrophy. Death from nephritis, marasmus, or pulmonary diseases is liable to supervene in these latter.

Treatment.—Warm clothing and every precaution necessary to guard the patient against exposure to cold should be insisted upon. Cod-liver oil, massage, warm baths, exercises, improved hygienic and dietetic measures calculated to raise the state of the general nutrition, are indicated. Thyroid-feeding is stated by several recent writers to have been followed by excellent effects; though the opposite result is also reported. Bertin recommends vaso-dilators, and most authorities agree that salicylates at times afford some amelioration; they are clearly indicated when rheumatic symptoms—viz. joint-pains—are prominent during the early phases of the disease.

Circumscribed Scleroderma, known also as Addison's Keloid and as *Morphœa*, is more common than the diffuse symmetrical form of the disease. It occurs more frequently in women than in men, and appears in unilateral irregular-shaped patches of various sizes, from that of a sixpence to a crown-piece, or much larger, without any symptom save slight itchiness. These patches may appear anywhere upon the body, but they are more commonly situated about the breasts or neck, sometimes following the course of a nerve, especially when seen on the limbs or face. The skin is of the colour of an old white marble statue; it feels smooth, firm, indurated and brawny, and usually can be pinched up, unless the plaque is very large and has contracted adhesions to the underlying tissues, which is rarely the case. The secretion of sweat is absent in the affected area, but the sensory functions are little, if at all, interfered with. The patches are but very slightly, or not at all, elevated above the level of the healthy skin, and in the atrophic variety of the disease they may be depressed; they are often marked at their borders by a zone of pale erythematous redness, in which dilated capillary vessels are visible. A patch nearly as large as the hand may develop within a few weeks, and may remain for many months or several years, one patch showing resolution as others slowly develop and increase in size by the coalescence of small atrophic areas, which become indurated as they join the primary plaque.

Occasionally, especially in childhood, the disease appears in the form of cicatricial bands or cords, somewhat resembling

in appearance the keloid scars of Alibert; but there are no claw-like processes and they are not vascular. This form sometimes affects the regions supplied by the fifth nerve and may involve the mucous surfaces, or it may extend either as a raised ridge or as a depressed sulcus, running in the direction of a limb for the greater portion of its length. Pigmentation often occurs in some portions of the patch, whilst other portions are of ivory-whiteness. Sometimes, but not often, the patches are symmetrical and numerous, being scattered over various regions; more usually they are confined to one part, as the neck, breast, or face.

The etiology and pathology of the disease are as obscure as those of the diffuse symmetrical form, but that the conditions are closely allied is rendered probable by the fact of their being found together (*mixed sclerodermia*) in the same subject. Thus a diffuse sclerodermia has been known to supervene upon a few isolated patches, and several circumscribed patches have been found on some portion of the face or neck where a considerable area of the body had been involved in a diffuse sclerodermia. Crocker found that the essential anatomical changes were due to increase in the connective and elastic tissues from fibrillation of the cells poured out around the small blood-vessels. When the arterial supply was entirely cut off an atrophic spot resulted; when this was partial, connective tissue hyperplasia, or true morphœa resulted.

Diagnosis.—*Leucodermia* is simulated in the atrophic cases where the pigment appears in patches on pale ivory-like areas; but the essential feature in leucodermia is the formation of a white patch in which the pigment is concentrated in the convex circumferential margins. The parchment-like induration of sclerodermia is never seen in leucodermia, as the skin in this latter affection always shows the normal suppleness.

Leprosy, when simulating circumscribed sclerodermia, is differentiated by the detection of anæsthesia and by the presence of the characteristic pigmentation, neuralgic pains, hyperæsthesia, and muscular atrophy which accompany the nervous form of the disease.

Crocker has drawn attention to the fact that some cases of *unilateral atrophy of the face* are examples of fifth-nerve morphœa which attacked the individual in childhood, causing wasting or arrest of development.

Circumscribed sclerodermia, which, as already stated, is sometimes known as the true keloid or cheloid of Addison, is not to be confounded with the *keloid of Alibert*, described upon page 750. This condition, even when not the result of an obvious scar-wound, is readily differentiated from circumscribed sclerodermia by the hard claw-like processes

which are usually present and by the vascularity of the new-growth.

Prognosis.—This is better than in the diffuse form, except where bands or cords exist, when resolution may not take place for many years, during which wasting of the underlying tissues occurs, leading to permanent deformity, as in the fifth-nerve examples of the disease. As a rule, the patches ultimately disappear, leaving the skin in a normal condition ; but the process of resolution may occupy twelve months, or nearly twice as many years.

Treatment.—The disease is uninfluenced by drugs given by the mouth, but the same measures are indicated as in the diffuse form. Of these, massage is of most importance. Electrolysis, employed as for nævoid growths, is sometimes followed by marked improvement. Many observers, including Neisser and Juliusberg, have reported success from the subcutaneous injection into the patches of from 5 to 10 mins. of a 10 per cent. solution of thiosinamin ; but in the hands of others this method has failed.

SCLEROSIS

(See under DISSEMINATED CEREBRO-SPINAL SCLEROSIS, p. 309 ; and LATERAL SCLEROSIS, p. 1227.)

SCROFULA

This is but a phase of tuberculosis, the term now being accepted as a synonym for tuberculosis of the lymphatic glands ; though still a few authorities adhere to the old view that scrofula and tubercle are not identical, but that tubercle is usually engrafted upon the scrofulous condition. In the articles upon 'Phthisis,' 'Tuberculosis,' 'Mesenteric Gland Disease,' 'Mediastinitis,' 'Peritonitis (Tubercular),' &c., the subject is already fully dealt with ; but the term is most commonly applied to tuberculosis of the lymphatic glands in the neighbourhood of the jaw and in the neck. The bacillus finds an entrance to the lymphatic system and is arrested in the glands ; during its sojourn there, owing to the less favourable conditions for growth than those which prevail in the lung, serous membranes, and bones, it often remains for an indefinite period in a quiescent state, and in the great majority of cases resolution occurs. Owing possibly to multiple infection, suppuration takes place in some cases ; probably in the majority of such examples the enlarged gland or glands are infected from a local or peripheral source, the tumour being practically a bubo. The portal through which the

tubercle bacillus usually finds its entrance into the cervical glands is generally regarded as the tonsil, since evidence of tonsillar tuberculosis has been abundantly demonstrated of late years. The frequency with which the condition is associated with carious teeth must not, however, be overlooked; but whether the infection in this case is direct or follows a secondary involvement of the tonsil, caused by the development of the bacillus in the first instance in the favourable nidus of the granulation-tissue of the diseased gums, remains to be demonstrated.

It is, in the writer's opinion, a common error to regard all cases of tubercular adenitis occurring in the cervical region as due to local infection. In discussing the pathogeny of tuberculosis and of phthisis the view that the tubercular infection occurs in the vast majority of instances through the intestinal epithelium has been dwelt upon. That this was a probable route has long appeared obvious to those who have studied the manifestations of tuberculosis in young subjects into whose dietary uncooked milk largely enters. The fact of pulmonary tuberculosis being produced by intestinal infection, even when the bacilli are inhaled, has been demonstrated by the epoch-marking experiments of Guérin and Calmette, referred to upon page 1301.

The writer has observed several outbreaks of tubercular adenitis distinctly traceable to the feeding of school-children with the milk of cows proved to be suffering from tuberculosis, and in some of these adenitis-cases mesenteric disease was also present. The invasion in these cases of the cervical glands by the bacilli is probably an accidental phenomenon, possibly due to mechanical or filtration processes.

The attenuation of the bacilli after their imprisonment in the lymphatic-gland structure has been demonstrated by inoculation-experiments upon guinea-pigs and rabbits, and it is, moreover, an obvious and well-recognised clinical fact that the great majority of cases of tubercular adenitis end in complete resolution. This is seen in its most striking form in the perfect recovery which often follows extreme enlargement of the mesenteric glands, even when associated with ascites and peritoneal effusion, without the aid of abdominal section.

Notwithstanding, however, the attenuation which the virus undoubtedly undergoes in the majority of instances, there is always a danger of a general blood-invasion should some anatomical change occur permitting a shower of the bacilli into the blood-stream. This is demonstrated by the fact that often in a fatal acute general tuberculosis old foci are discovered in some lymphatic centre. This fact lends considerable support to the practice, first suggested by Clifford Allbutt, of excising these dangerous cervical enlarged glands.

The writer has witnessed the most permanent and satisfactory results from this method, but only in those examples of chronic tubercular adenitis of a circumscribed character, and notably in a case where a large indurated tubercular tumour, consisting of a mass of lymphatic glands, had existed for several years in the axilla of a patient seen also by Sir Clifford Allbutt. The excisions of whole chains of lymphatic glands the seat of tubercle, as carried out by modern surgeons, not unfrequently are followed by phthisis or other visceral form of tuberculosis. It is a well-recognised fact that partial operations upon tuberculosis of bone are fraught with great danger of a general infection, and the writer has witnessed this result in at least a dozen cases of cervical adenitis after the most extreme care upon the surgeon's part in removing every gland exposed during a prolonged operation.

SCROFULODERMIA

This is a condition often associated with the disease described in the preceding article, and closely allied to lupus vulgaris, since it consists in an invasion of the skin by the tubercle bacillus.

Clinically, scrofuloderma can often be differentiated from lupus, but for all practical purposes, including pathology, diagnosis, prognosis, and therapeutics, the two diseases may be regarded as identical; moreover, they very frequently are associated together in the affected region.

The disease is seen in its most typical form where a tubercular lymphatic gland has broken down, involving the cutaneous covering lying directly above it. As the cascating gland softens the skin becomes reddened and ulcerates, and the bacilli invade it through the sinuses leading down to the lymphatic tissue discharging the tubercular organisms into the layers of the cutaneous tissue exposed in the thin undermined ulcerated edges. In this manner a slowly extending strumous ulceration, characterised by punched-out but thin undermined edges, may invade a considerable area of the skin in the neighbourhood of the gland. In other cases, as pointed out by Crocker, true lupus may originate in the immediate neighbourhood of a sinus, with its highly characteristic nodules and slowly infiltrating features, contrasting markedly with the more actively destructive process of scrofuloderma, or both processes may be seen progressing side by side.

Occasionally scrofuloderma arises independent of adenitis, as in strumous disease of the digits; in which case it may start in a tubercular nodule in the subcutaneous tissue, subsequently involving the bone; or, originating in the osseous

tissue, which necroses, it may afterwards invade the skin, as in the lymphatic-gland cases.

The treatment is mainly surgical in all cases, consisting of free excision, followed by scraping and the application of any of the corrosive or antiseptic applications indicated in lupus.

SCURVY, or SCORBUTUS

This is a blood-disease, characterised by profound debility, sponginess or ulceration of the gums, anæmia, cutaneous petechiæ, and brawny swellings, the result of hæmorrhagic infiltration, the mucous surfaces or internal organs sometimes also being involved. Two forms of the disease are recognised—the scurvy of adults and of infants.

SCURVY OF ADULTS

This, formerly one of the most common, has now, owing to the universal attention to preventive measures, become one of the rarest of diseases. It is still, however, endemic, and occasionally epidemic, in some parts of European and Asiatic Russia. Formerly its outbreaks were numerous in navies and in the mercantile marine services in the days of the sailing-ship, but land-scurvy is seldom seen, except in times of war or famine. It occurs in all climates, affects all races, and both sexes, at all ages.

Etiology.—The view was formerly accepted without reservation, and is still adhered to by many, that the disease is the direct result of the absence of fresh vegetables from the diet. This opinion seemed to be true beyond question from the time that a ration of fresh lime-juice was introduced into the dietary of sailors in the British Navy after the threatened disasters to Lord Howe's fleet in 1795. Since that period the disease has rapidly declined, and upon the compulsory introduction of a similar measure in the mercantile marine seventy-two years later the disease almost entirely disappeared. Various speculations have always been indulged in with the view of explaining or determining the specific agent whose presence prevented the disease; thus Garrod maintained that the condition was due to the absence of potash-salts, whilst other authorities maintained that the vegetable-acids were the effective agents.

These views, however, must be seriously modified by the consideration of the fact that scurvy practically never occurs in children fed upon the breast or upon an exclusive diet of pure fresh milk, and that the disease seldom, if ever, appears in adults who have lived for considerable periods upon a diet consisting entirely of fresh animal food, as proved by the experience of Arctic explorers. This latter fact is very

important, since outbreaks of scurvy have been known under identical hygienic surroundings in Arctic travellers who have lived upon tinned meats and canned vegetables, and in whom the symptoms of scurvy disappeared upon resorting to the fresh meat of the seal or bear. It is therefore conclusive that some ingredient also exists in animal food which prevents scurvy, and it is equally certain that this constituent is destroyed in the process of preserving the food. This is further demonstrated by the fact that infantile scurvy is practically never seen in children fed exclusively upon fresh milk, but that the disease is common amongst those who are brought up upon preserved or sterilised milk or upon patent foods containing milk which has been desiccated, as pointed out by Colman.

The consideration of these facts has led to the theory that scurvy is due to a toxæmia caused by the production of some ptomaine or other poison generated in the food. This view is rendered feasible by the results of those who have been able to produce an allied, if not an identical, condition in monkeys by feeding them upon tainted or partially decomposing food, both vegetable and animal. Upon the other hand, it appears extremely difficult to apply the theory of a toxæmia, in the ordinary sense in which the term is employed, to those cases in which scurvy has been caused by a diet of tinned foods in an apparently excellent condition of preservation. It would seem necessary to shift the theory of toxæmia in such cases from the supposed contamination of the food-supply to the intestinal tract. If it be assumed that the preparation necessary to ensure the preservation of the food or milk entails such changes in its composition that intestinal disturbances are produced, then ptomaine-toxæmia may arise from decomposition of the food within the body.

It must not, however, be forgotten that only a small percentage of the individuals fed upon preserved or sterilised foods become scorbutic: hence the theory that some other positive etiological factor is at work; and of late years the view is largely held that the disease is of microbic origin. That some such theory is necessary is obvious from the absence of scurvy in professional 'fasters,' since in their case both fresh and preserved food and fresh vegetable-juices are absent for long periods from the alimentary tract. Saito and Okada have recently claimed that they have isolated the specific micrococcus from the blood of scorbutic patients. Ausset also maintains that infantile scurvy is due to an infective process of which he believes the micro-organism described by Lewine under the name of the *bacillus hæmosepticus hominis* is the cause. Wright regards scurvy as an acid intoxication of the blood.

If these researches are corroborated, a working hypothesis will be available which clears up all the etiological factors

hitherto recognised as playing a rôle in the production of the disease. Thus the presence of some constituent in fresh vegetable-juices and in fresh animal food may be conceived as inimical to the growth of the microbe; when this constituent is absent the micro-organisms may be supposed to develop and manufacture their toxins unchecked.

Though the disease may occur at any age, it is most severe in old subjects whose vital resistance is reduced. Formerly the fact that males were much more frequently affected than females was explained by the preponderance of males subjected to the exclusive dietary prevailing in ships and armies; but in sieges the much larger proportion of males affected can no longer be denied. Probably the explanation of this sexual selection may be explained by a fact observed in scurvy occurring in South Africa by Neil Macvicar, who found that muscular exertion precipitated the onset of the disease. He observed that natives living in their homes on a diet deficient in either fresh meat or vegetables often showed no signs of the disease till they undertook some work entailing active muscular exertion. In a similar manner overcrowding, depressing mental exhaustion, with or without physical fatigue, are recognised as predisposing causes.

It may be accepted, therefore, that with abundance of fresh animal food lime-juice is not absolutely necessary as a prophylactic, even in the absence of fresh vegetables; and it is probable that a considerable diminution in the frequency of the disease may be fairly attributed to the advent of steam as a propulsive agent, since fresh food is so easily procurable throughout all modern voyages. In the few cases where the disease has occurred in spite of a lime-juice ration it is possible that the prophylactic had undergone some change akin to what occurs in sterilising fresh milk, or else the microbic infection had been unusually severe in such cases.

Symptoms.—The onset is slow and insidious; gradual loss of strength, both mental and physical, progresses along with anæmia till a distinct degree of cachexia becomes apparent. Though the conjunctivæ are pearly-white, the colour of the skin is sallow or earthy; to the touch it becomes dry and harsh; petechiæ soon appear, especially in the immediate neighbourhood of the hair-follicles; and large ecchymoses develop upon the limbs, usually starting on the legs and spreading to the arms and trunk. These hæmorrhagic extravasations are precipitated by the slightest contusions, frictions, or even scratchings.

The gums are affected early, and their condition is of great diagnostic importance. The first sign consists in a swelling or sponginess of the projections between the teeth, associated with lividity; bleeding and ulceration follow, or sloughing occurs in severe cases, and the breath becomes very foul,

Ecchymoses are common on the mucous membrane lining the inside of the mouth, but ulceration here does not occur; the tongue swells, and as the changes in the gums progress the teeth loosen and often drop out, and in very severe and neglected cases the alveoli necrose, especially when previous dental caries has existed. The sponginess may be entirely absent in those portions of the gums where teeth do not exist, and it is often unobservable in edentulous patients, and is always aggravated in the vicinity of carious teeth. When ulceration occurs, the swollen gums may project in livid fleshy masses beyond the pallid lips. In some rare cases, however, the gums have been observed to remain pallid, smooth, and unswollen throughout, even when the other signs of the disease are present in an aggravated form. The eyelids are oedematous and often livid; sometimes petechiæ occur upon the conjunctiva, and pitting of the ankles is commonly present.

With the progressive development of the ecchymoses and petechiæ in various regions there often appear large brawny swellings, especially upon the legs, popliteal spaces, and thighs. These consist of extensive hæmorrhagic infiltrations, in which the hamstring and calf muscles may be entirely encased, and the muscles themselves are found the seat of numerous extravasations, whilst the subcutaneous tissues are soaked in a blood-stained fibrinous fluid. A somewhat similar feature is the periosteal swellings or nodes which often appear upon the tibiæ, jaws, or other bones; these are due to extravasations of blood between the periosteum and the bone, and form a highly characteristic feature in the infantile form of scurvy. Hæmorrhages from the mucous surfaces are not rare, epistaxis being the most common; but hæmatemesis, hæmaturia, and melæna also are sometimes observed, chiefly as the result of complications, the latter symptom being usually due to dysenteric ulceration. Infarctions in the spleen, kidney, and lung are a feature in some cases, but the brain usually escapes.

With these hæmorrhagic extravasations the debility steadily increases and the cardiac power fails, so that breathlessness, which is often an early symptom, may become extreme, and is liable to be associated with attacks of syncope. The mental condition is one of apathy, complete listlessness, or torpor, sometimes associated with intense headaches and rarely with convulsions. A very interesting symptom in some cases to be met with is the hemeralopia or nyctalopia referred to upon page 1147. This is due to the early exhaustion of the retina when exposed to bright light, and is unattended by ophthalmoscopic changes.

Muscular pains are often complained of throughout the disease; those felt in the legs are greatly exaggerated by the

presence of the brawny swellings, and joint-pains are very commonly severe. In former times necrosis of bones, disintegration of joints, and even separation of epiphyses were not uncommon in neglected cases. There is a marked tendency towards ulceration or even sloughing of the skin over extravasated areas, and old scars and recent fractures are liable to break down. The scorbutic ulcers are prone to spread, leaving fungoid sores with punched-out edges after the separation of the dry black eschars, which exude a thin sanguineous ichor. The temperature may remain normal or sub-normal throughout, but the advent of complications usually is ushered in with a moderate degree of pyrexia. The most frequently observed complications are dysentery, pleurisy following extravasations of blood into the pleural sac, pericarditis from similar causes, pneumonia, or gangrene of the lung. Dropsy is not uncommon, and may be associated with a considerable amount of albuminuria; but, upon the whole, renal mischief is not common, and hæmaturia is certainly less frequent than in purpura. Difference of opinion exists about the state of the urine as regards the proportion of solids; though the specific gravity is increased, the alkaline phosphates appear to be often diminished in amount.

The progress of the disease towards a fatal issue is now very seldom witnessed, since its causation and treatment are more clearly appreciated. Rarely is the extreme emaciation which is a prominent feature in the advanced stage of the disease seen nowadays. When death occurs, its usual immediate cause is syncope—part of the general asthenia; but this is unlikely to occur except in patients weakened by some previous disease, as malaria or syphilis, or reduced by any of the complications, as dysentery, pneumonia, pleural effusion, pulmonary œdema, or pericardial extravasation.

Morbid Anatomy.—The main changes are those incidentally mentioned in the narration of the symptoms—viz. the presence of the hæmorrhages into the skin, beneath the periosteum, surrounding the muscular sheaths and within the muscular substance, into the serous cavities, into the joints, and occasionally also in the lungs, kidney, and spleen, and even within the bone-substance. More or less fatty change is observed in the heart, muscles, and internal organs, such as occurs in all cachectic conditions. There is little or no wasting of the subcutaneous fatty tissue, except in cases of protracted duration, which are now never seen. The blood, contrary to expectation, shows little change. Older observers maintained that its fibrin was in excess; Wright found that its alkalinity was constantly diminished in the Netley cases. The discovery of the micro-organism recently described by Okada and Saito has already been referred to. Jackson and Harley, in the experimental scurvy of monkeys induced by

feeding the animals upon tainted foods, found marked chlorosis, leucocytosis, and slight increase in the fibrin.

Diagnosis.—There can seldom be a difficulty in the recognition of the disease when it occurs, as it usually does, in numbers of individuals subjected to the deprivation of fresh food. When the affection appears in an isolated case it is, however, liable to be mistaken for *purpura*, especially should the gums show no sponginess. The diagnosis under these conditions may be impossible unless the former dietary of the patient has been scrupulously investigated. The discovery of the absence of fresh animal and vegetable food in the diet, together with the presence of marked debility and cachexia, and multiple extravasations into the muscles, bones, and subcutaneous tissues, usually settles the diagnosis; but should doubt remain, it is speedily dissipated by the administration of fresh vegetable-juices, which cause almost immediate amelioration of all the symptoms. When sponginess of the gums exists, which is, as already stated, nearly always the case, there is no difficulty in eliminating *Henoch's purpura*, *Schönlein's disease*, or *Werlhof's purpura*, since in these affections the characteristic gum-condition of scurvy is always absent, and there is no history of deprivation of fresh food, and early cachexia is not obvious. It should always be borne in mind that, though hæmorrhage from the mucous membranes does occur sometimes in scurvy, it is never a prominent clinical feature as in Werlhof's purpura, and in this condition anæmia is not marked, unless after prolonged bleeding; whilst in scurvy the anæmic condition ushers in the disease.

Leucocythæmia is easily differentiated by the splenic or lymphatic enlargements and by the characteristic leucocytosis, even should the gums show any thickening or tendency to bleed. *Hæmophilia* is recognised by its remarkable hereditary history, by the evidence of the hæmorrhages being due to traumatic causes, and by the absence of cachexia. *Pernicious anæmia* is differentiated by the blood-characters and by its long duration. *Splenic anæmia* is not so easily excluded, since the blood-changes are usually negative; but the great enlargement of the organ, with its tenderness and attacks of severe pain and moderate pyrexia, are more or less characteristic, though bleeding from the gums is often present. In the exclusion of all these last-mentioned diseases, characterised as they often are by hæmorrhagic tendencies, it is hardly necessary to point out that the deprivation of fresh animal or vegetable food plays no part in their history or causation, and the other characteristics of scurvy are wanting, the progressive anæmia and debility excepted.

Prognosis.—The rate of mortality was very high in former times; now it may almost be regarded as *nil*. Only in long-neglected cases is there practically any danger to life

to be expected, as the disease responds so speedily to proper dietetic treatment.

Prophylaxis.—Nothing further need be said regarding the prevention of scurvy, since the subject has been fully discussed under Etiology. The occurrence of the disease is practically impossible save in the absence of fresh animal or vegetable food; and where these cannot be procured, as in long voyages, the administration of fresh lime or lemon juice acts as an efficient prophylactic. Citric and tartaric acids are valueless.

Treatment.—This is mainly dietetic. Fresh vegetable-juices, salad, potatoes, and fruits should be given freely as soon as the nature of the malady has been recognised, and these should be followed by fresh juice expressed from raw meat; and later on, as soon as mastication is possible, fresh underdone beef-steak or roast meat may be allowed. The best routine treatment for a scurvy-stricken ship's crew upon admission to hospital would be a generous diet of beef-broth in which an abundance of fresh vegetables has been boiled for a few minutes before straining and serving up. No other treatment is necessary in uncomplicated cases, as no drug (save fresh lime or lemon juice) has any influence upon the disease. Hæmorrhages, if not speedily checked by the altered dietary, should be treated by chloride of calcium internally, and other complications met by recognised therapeutic agents. Syncope must be guarded against by keeping the patient in the recumbent posture.

SCURVY OF INFANTS

Infantile scorbutus, Barlow's Disease, or, as it is sometimes called, Scurvy Rickets, differs in no essential from the scurvy of adults.

Etiology and Pathology.—This has been discussed incidentally in considering the causation of the adult form of the disease, but the conditions under which infantile scurvy is produced necessitate a brief repetition. The disease is a comparatively modern one, and, as pointed out by Colman, it is a product of over-civilisation. It practically never shows itself before six months after birth and very rarely after eighteen months, most of the cases occurring in infants of about one year old. Entirely breast-fed infants never suffer from the disease, except in the rare cases where the mother is already scorbutic from the deprivation of fresh animal or vegetable food. The natural food of infants is fresh milk, and infantile scurvy never occurs when the child is exclusively fed upon it. Cases are very occasionally seen in which fresh milk has been the chief dietary, but always some farinaceous element has been also administered. There can be no doubt that the almost invariable etiological factor

present is a diet in which prolonged sterilisation, peptonisation, condensation, or desiccation of the milk has destroyed its antiscorbutic properties. 'Humanised' milk and all the numerous patent foods are liable to produce the condition. When, however, this is fully recognised, there remains the obvious fact that but a small proportion of infants exclusively fed upon such food become scorbutic. To explain this difficulty some authorities maintain that another etiological factor must be present, and this is surmised by Colman to be due to a toxic action, the result of some decomposition in the food which does not necessarily render it offensive to taste or smell. Ausset, as already stated, regards the disease as the product of infection absorbed from the alimentary tract, the toxin being probably manufactured there by Lewine's bacillus.

Infantile scurvy was formerly regarded as a variety of rickets, but this view is now abandoned; true rickets certainly sometimes accompanies it, but the diseases are essentially different. It is a noteworthy fact that rickets is commonly a disease of the poor, and infantile scurvy affects most frequently the children of the better-class individuals, because, as pointed out by Cheadle, scurvy-producing patent foods are beyond the reach of the poor; he also draws attention to a fact which cannot be questioned—viz. that the children of the poor are treated at an early age to potatoes and other antiscorbutic articles of diet. Another explanation is also available: the mere bringing of the milk to the boiling-point does not appear to very materially minimise its antiscorbutic power; and this is the usual practice amongst the poorer class if they apply any heat at all in the preparation of the infant's food, but the craze amongst the better classes of destroying all forms of microbes often exhibits itself in prolonged sterilisation of the infant's pabulum.

Symptoms.—These, in the main, when the disease has fully developed, correspond with the symptoms observed in the adult form, as first clearly pointed out by Barlow and Cheadle, who differentiated the affection from rickets. The onset is insidious, as a rule; there are pallor, muscular debility from flabbiness, and loss of tone; but the first obvious symptom to draw attention to the condition may set in with a considerable degree of abruptness. This symptom, which is very characteristic, in most instances shows itself in marked tenderness and pain in the lower extremities. The child screams when the legs are moved by the nurse, as in lifting it from its cot, in dressing or bathing, though when left to itself it lies quiet and peaceful, with the legs drawn up and motionless. At a later stage, when the muscular debility and flaccidity have progressed, the child lies with its lower extremities everted and apparently paralysed, as in a case of polio-

myelitis, but so great is the tenderness that screaming is easily elicited even by the approach of the nurse or attendant, owing to the dread of being touched or disturbed. At this stage the upper limbs show no signs of tenderness, but careful examination may nevertheless sometimes detect slight signs of the rachitic changes which so commonly accompany the disease. Thus some swellings may be found about the epiphyses, and beadings may be observed on the ribs.

Palpation will reveal upon the legs the most characteristic lesion of the disease, which consists in swellings upon the tibiæ above the ankles, caused by hæmorrhages underneath the periosteum. The membrane may even be detached entirely from the underlying shafts of the bones by a thick layer of blood-clot, as first pointed out by Barlow; these lesions are roughly symmetrical, both tibiæ being involved, and often the shafts of the femora suffer similarly. These periosteal swellings are obviously the cause of the great tenderness of the limbs, and at a later stage may affect the bones of the fore-arm above the wrists, the scapulæ, humeri, skull, or facial bones, but always to a slighter extent than the tibiæ. Hæmorrhages at the same time occur into the subcutaneous tissues and into the muscles, forming swellings which correspond to the brawny indurations met with in the adult disease, and the legs become oedematous.

Fractures of the bone are not rare. These are liable to occur anywhere in the shaft, but separation of the epiphyses is more common, especially at the upper end of the tibia or at both ends of the femur. The joints themselves remain free, though sometimes the accompanying rachitic enlargement of the epiphyses may closely simulate arthritic mischief. In some cases several ribs on each side fracture close to their costal cartilages, producing a remarkable depression of the sternum and adjoining cartilages, which appear as if driven inwards towards the spinal column.

Unless the hæmorrhagic extravasations are great and the tension high, there is no pyrexia; but the anæmia steadily increases, the skin becoming sallow. The muscular debility progresses and the child is unable to sit up owing to extreme weakness of the lumbar muscles, standing being impossible from the first owing to the tenderness in the lower extremities. Hæmaturia is a very common sign, and epistaxis is not rare; bleeding from the bowel may be also present. The most remarkable hæmorrhagic sign, however, is that which occurs under the periosteal lining of the bones entering into the formation of the orbit. This may occur suddenly, causing proptosis or protrusion of the globe, often accompanied by ecchymosis of the conjunctiva and œdema of the eyelid, the involvement of the opposite eye following in a day or two. Snow states that this occurs in about one-seventh of all cases,

Ecchymoses appear upon the slightest injury to the skin, and occasionally small true purpuric vibices are visible.

The condition of the gums is important, but if teeth have not already appeared the gum-tissue may appear normal. Where the eruption of the teeth has already been accomplished the gum-tissue is swollen, spongy, livid, and soon shows the tendency manifest in adult scurvy to ulcerate, bleed, and project beyond the lips in fungating, foul, plum-coloured masses, and the teeth may be shed. Unless the condition of the mouth is so severe as to prevent feeding, emaciation seldom becomes a marked feature, even when the anæmia is profound; but cardiac asthenia is usually obvious, as seen in the tendency towards syncopal attacks.

Minor degrees of infantile scurvy are common, and are liable to be overlooked, the only symptoms present being some degree of anæmia, hæmaturia, and general tenderness of the limbs, with slight swelling of the gums over teeth about to force their way through the livid tissue. Cheadle has pointed out the fact that proptosis may be the only obvious clinical feature in some cases, whilst others have reported cases where the only symptom present was hæmaturia, which immediately disappeared after the administration of fresh vegetable-juice.

Morbid Anatomy.—The changes correspond with those observed in adult scurvy, as first pointed out by Barlow. The graver involvement of the periosteum and the epiphysial fractures are probably due to the greater activity in the osteal and periosteal development during infant life; and this may be the explanation of the fragile state of the capillary-walls also observed in the disease. The only essential difference between the two conditions, if it can truly be regarded as essential, is the frequency with which evidence of rachitic changes are found in the epiphyses in the infantile form; but these are usually slighter than in ordinary rickets, and in a considerable percentage of cases are entirely absent.

Diagnosis.—This is never difficult when the gums are involved, as they always are in children whose teeth have appeared. The clinical picture of a progressive anæmia and cachexia without obvious wasting and the marked tenderness of the lower extremities, causing screaming upon the slightest forced movement in a child who lies when undisturbed peacefully resting with the legs flaccid and everted, cannot be mistaken, especially when periosteal swellings are visible or palpable upon the tibiæ above the ankle-joints.

Infantile paralysis, or *paraplegia*, is easily excluded by observing that the loss of power is mainly inhibitory; the affection is symmetrical, whilst in poliomyelitis it is usually unilateral; there is no fever at the onset, wasting of the muscles does not follow, and the electrical reaction is normal,

Acute rheumatism is excluded by the apyrexia, by the localisation of the pain and tenderness, which is never confined to the joints; these are also never the seat of redness, heat, or swelling.

Hip-joint disease is excluded by its own characteristic signs and slow development, and by the fact that the pain and tenderness in infantile scurvy exists in both lower extremities.

Syphilitic epiphysitis, though associated with pain and apparent loss of power in the affected limbs, almost always occurs before the sixth month, and other evidence of syphilis in the infant and in its history are forthcoming.

Acute osteomyelitis or *acute necrosis* and *periostitis*, are excluded by the absence of severe constitutional symptoms, as high fever, shivering, sweating, &c., and by the symmetrical or bilateral distribution always present in the scorbutic affection. The fever in infantile scurvy is, when present, of a mild and transient nature, and only occurs, as already stated, when the periosteal extravasations are extensive and associated with considerable tension—a condition which tends to pass off as the membrane becomes elevated from the underlying shaft of the bone.

Fractures due to infantile scurvy are liable to be mistaken for the result of simple traumata, but such a mistake is easily rectified by an examination of the periosteal swellings and gums.

Stomatitis is simulated when the ulcerated condition of the gums forms the predominant feature of the case; but this affection is eliminated by a careful examination of the mouth, which shows no ulceration save in the region of the gum-tissue, whilst in both the *ulcerative* and *gangrenous* forms of stomatitis the interior of the cheeks are involved.

Rickets.—As this condition is often present there is a danger of a case of infantile scurvy being regarded as one of simple rickets, but the difficulty is seldom great. True rickets shows no tendency towards hæmorrhagic extravasations—periosteal, muscular, subcutaneous, nephritic, intestinal, or otherwise—and when epiphysial, costal, or cranial changes are present as a complication of infantile scurvy they are always slight, laryngismus, convulsions, and head-sweats being absent. In doubtful cases (and this remark applies to the diagnosis of all the allied conditions) the result of anti-scorbutic treatment is emphatic and final, true infantile scurvy rapidly responding, whilst all other affections, including the rachitic, fail to improve.

The exclusion of *hæmophilia*, *purpura hæmorrhagica*, *leukæmia*, or *lymphadenoma* is determined upon after a consideration of the differential points mentioned under the Diagnosis of adult scurvy.

Prognosis.—Only in neglected cases or where the disease has been mistaken for some other affection is death likely to occur. The progress of the affection when uninfluenced by treatment is probably always towards a fatal issue, death being due to hæmorrhage, syncope, some pulmonary complication, as bronchopneumonia, or it may be the result of a general progressive marasmus or asthenia. Probably, as the result of some happy accident determining an alteration in the dietary, recovery does follow in unrecognised cases. There is very rarely any permanent deformity left behind once the patient's recovery is made certain by proper dieting, but occasionally fractures may set in an abnormal position.

Treatment.—As regards *prophylaxis* no further comment is necessary beyond those included under Etiology, the disease being invariably due to errors in dietary caused by the destruction of the antiscorbutic quality of fresh milk by prolonged sterilisation, concentration, desiccation, or peptonisation, or to the exclusive feeding of the infant by patent foods or purely farinaceous substances.

When the disease has become established, however, it is not sufficient to trust alone to the antiscorbutic action of fresh milk. If cow's milk cannot be digested by the infant, the services of a healthy wet-nurse are imperatively demanded, and even then some fresh vegetable-juices or raw meat-juice should be also administered. In older children the plan recommended by Cheadle and by Colman is valuable; it consists in adding potatoes to the fresh-milk dietary. This vegetable, after being cooked without previous peeling, is passed through a fine sieve and added in small quantity to the milk. Raw meat-juice is decidedly valuable, but the writer prefers to give a large piece of almost uncooked tender beef-steak into the care of a reliable nurse, who firmly holds one end of it in her fingers, allowing the child to suck the other. Lemon-juice and orange-juice should also be given in small quantities, or fresh vegetables may be added to beef-tea, mutton-broth, or chicken-soup near the end of the cooking-process, and the mixture passed through a fine sieve.

The improvement is surprisingly rapid after the commencement of the fresh vegetable-juice diet; sometimes within forty-eight—Colman states within twenty-four—hours the acuteness of the pain and tenderness passes off.

SEA-SICKNESS

This condition is sufficiently defined by its name, and the symptoms require no description. The theories invented to explain the phenomenon of sea-sickness are innumerable, but no really satisfactory explanation is yet forthcoming;

it is therefore unnecessary to discuss the evidence in favour of such hypotheses as labyrinthine and visual disturbances, anæmia, concussion, or hyperæmia of the brain, spasmodic closure of the glottis or pylorus, altered innervation affecting the sympathetic ganglia in the abdomen, &c. &c.

If some attempt must be made to classify the malady, the best is to place it in the category of 'cerebral vomiting,' as done by Purves Stewart, always, however, remembering that, though the main symptoms are nausea, vomiting, and retching, more or less vertigo, giddiness, and often cephalalgia are also present. The obvious etiological factor is the peculiar mechanical movements of the ship, either of the rolling or pitching kind, or a combination of both of these motions, which excite the functional disturbance of the stomach and derange the functions of the semicircular canals. The predisposing causes are often psychical; neurotic subjects suffer most frequently and severely, and in them the accompanying smells, noises, and vibrations inseparable from the environment of steamships are powerfully acting secondary factors. The writer has witnessed a severe attack induced in a neurotic subject as soon as she had entered a stuffy cabin, before the ship's movements had commenced.

The remedies suggested as preventives or cures are even more numerous than the theories invented to explain the pathogeny of the malady. These are discussed in detail in the author's 'Dictionary of Treatment' (4th ed.), pp. 875 and 876. The best routine is to administer bromides in full doses for a couple of days before embarking, to wash out the rectum by a large enema, to take a light meal two or three hours previous to going on board, and to lie down flat upon the back, remaining quiet, with the head low and the eyes closed, after swallowing a pilule containing $\frac{1}{16}$ gr. of hydrobromide of hyoscyamine. Some temporary relief is usually obtained by the patient taking a deep inspiration each time that the ship makes a descent in the water. In the great majority of instances the condition tends to slowly pass off after about the third day, unless the weather remain boisterous, but upon long voyages relapses are common should the ship's movements become exaggerated.

The symptoms of collapse, fainting, &c., in protracted cases are to be met by stimulants administered in combination with effervescent water.

SEBORRŒA

This is a cutaneous affection characterised by the accumulation of the increased and altered secretion of the sebaceous glands, which either remains on the skin as an oily coating or separates in scaly films or masses.

The affection is also known as Stearrhœa and Steatorrhœa, and two well-marked forms of the disease are recognised; both usually commence in the scalp, tend to spread to the face, and sometimes invade the trunk.

Etiology.—The disease may appear at any age, but it is more common at puberty, and often occurs in otherwise healthy and robust subjects, though, upon the whole, depressing causes are often found in association with its advent, as gastro-intestinal disturbances, an attack of zymotic disease, the approach of the climacteric period, or, at an earlier age, chlorosis. The question of a microbic origin of the condition will be discussed in the section following.

Pathology.—Various widely divergent views are held upon this subject. Some authorities, especially Unna, maintain that the condition is due to disease of the sweat-glands, whose function is the manufacture not only of watery secretion, but of oleic acid, and the increase of the latter he holds as the explanation of seborrhœa oleosa. Sabouraud, on the other hand, insists upon true seborrhœa being the result of a fine bacillus flourishing in the mouths of the sebaceous glands, which, by stimulating their secretory cells, leads to the outpouring of a more fluid fatty secretion, causing seborrhœa oleosa. This latter authority regards seborrhœa sicca, commonly known as dandriff, as a distinct disease, to which he applies the term ‘pityriasis.’ Unna insists that seborrhœa sicca is the typical form of seborrhœa, the scales being produced from the horny epidermis by a dry catarrhal or inflammatory process induced by the presence of the morococcus organism, the scales being at a later stage mixed with fat. The difficulty in determining the importance of any organism as a causal agent in seborrhœa is obvious from the results of Taenzer, who isolated no fewer than eighty varieties of organisms from the seborrhœic secretions and scales.

Seborrhœa oleosa is the form of the disease in which the secretion of the sebaceous glands (or sweat-glands?) appears as a greasy or more or less fluid oily coating on the skin. It usually commences in the scalp, but owing to the presence of the hair its characteristics are less evident than upon the forehead and alæ of the nose, in which latter situation it is very commonly associated with acne and rosacea. Spreading from the scalp, in the first instance, to the face, the disease sometimes invades the trunk and limbs; it is extremely doubtful if it is ever found in the latter situations without some scalp-scaliness or oleorrhœa being also present. As the oleaginous material dries, scales of yellowish-brown colour form, and occasionally underneath these there is some redness of the skin. Behind the ears, in the arm-pits, popliteal spaces, groins, and upon the genitals the typical oily seborrhœa is often associated with eczema, forming a distinct weeping

eruption. This corresponds, in the main, to the condition described by Unna as *seborrhœic eczema*, in which he holds that the morococcus transferred from the scalp excites a true dermatitis around the sudoriparous or coil-glands. Crocker, whilst slow to acknowledge any true inflammatory process in ordinary seborrhœa, recognises an *eczematiform* seborrhœa or *seborrhœide*, in which an apparently true eczema supervenes upon the original seborrhœa of the sebaceous glands in the scalp and face. These forms of eruption are included by the French school amongst a group designated by them the 'Seborrhœides.'

Seborrhœa sicca constitutes the variety of the disease in which the fatty matter appears in an almost solid form, usually upon the scalp, which becomes covered with dry greasy scales, films, or dirty-looking yellowish-brown masses or crusts, consisting of fat and epithelium. In the infant these yellowish crusts are very common about the anterior fontanelle when washing is neglected; they appear to be of the same nature as the accumulation of secretion about the clitoris and prepuce; upon the scalp and elsewhere, as already stated, Unna maintains that they are the result of an excess of the secretion of the sweat-glands.

The most common form of dry seborrhœa is that long recognised under the name of *dandriff*, in which a fine, greasy, furfuraceous powder covers the entire scalp; the minute silvery scales fall off, and usually appear in evidence upon the dark clothing around the neck and shoulders. The divergent views of Unna and Sabouraud regarding this condition are epitomised under the Pathology of seborrhœa, upon the preceding page.

Whichever view be accepted, the fact remains that the condition often leads to thinning of the hair and permanent baldness (see under 'Alopecia,' p. 30). Sometimes a considerable degree of itchiness prevails, though usually the scalp, when the scales are removed, is found free from hyperæmia; and a similar condition may be observed upon the eyebrows and hairy parts of the face. The trunk and limbs may be also involved, the disease being supposed to spread by the transference of the infected scales by direct contact or transplantation by the finger-nails in scratching. As in the oily form, dry seborrhœa of the scalp may be found associated with eczematoid eruptions about the genitals or other skin-regions the seat of much moisture, which affords a suitable nidus for the growth of the parasitic organisms.

Diagnosis.—In the dry portions of the skin the eruption may closely simulate *psoriasis*, but the distinct greasiness of the finer scales, which lack the silvery lustre of psoriasis, and the characteristic feature of the eruption, which always commences in the scalp and extends gradually downwards

generally prevent errors in diagnosis. Moreover, the red hyperæmic base of psoriasis is distinctive. In this connection the recent contributions of Brocq are interesting. The French school regard all the cutaneous affections, from pityriasis to psoriasis, as belonging to one group, and Brocq describes a seborrhœa psoriasiformis as one of the connecting links. Under the term 'seborrhœides,' as already stated, most authorities include Unna's seborrhœic eczema, psoriasiform seborrhœa, and lichenoid or papular seborrhœa. Other dermatologists go further and embrace in the group pityriasis and acne vulgaris, since Sabouraud maintains that the latter disease (acne) is but a form of seborrhœa in which the mouth of the sebaceous gland becomes plugged by a horny concretion.

When seborrhœa attacks the region of the skin between the shoulders and over the front of the chest, and even in the groins, the eruption sometimes presents ringed or circinate characters, which are liable to closely simulate *ringworm* of the body and *tinea versicolor*; but the examination of the scalp prevents an error in diagnosis, since the characteristics of seborrhœa sicca easily exclude *tinea capitis*. This is the form of seborrhœa known as *S. papulosa*, *S. corporis*, or *lichen circinatus*, its chief characteristic feature being the groups of red papules, which as they join into patches spread at their margins whilst fading in the centre. The scalp is always affected along with the body.

Eczema is easily differentiated from most cases of seborrhœa, but the scaly stage of an old dry eczema closely resembles the dry form of seborrhœa. The chief distinguishing point is the fatty or greasy nature of the seborrhœic scales or crusts; these, on removal, exhibit usually a pale or normally coloured cutaneous base, whilst in eczema the base is distinctly hyperæmic and usually moist after scratching, and itching is a constant symptom. Moreover, in seborrhœa the entire scalp is usually involved, contrasting generally with the patchy distribution of eczema. When both conditions are present, as in Unna's seborrhœic eczema, the history and progress of the affection clear up any doubt.

Prognosis.—The disease, unless treated in the most persevering manner, tends to become chronic and incurable. The only element of importance in the remote outlook is the great danger of permanent baldness. This so frequently results that most authorities regard dry seborrhœa of the scalp as another name for the alopecia of middle-life; the polished, shining, bare scalp of premature senility being due to the excess of the sebaceous material which is the characteristic of the initial process. In young subjects, and especially in the infantile form, the prognosis is always most favourable.

Treatment.—Local measures only are of any value, and since the universal acceptance of a parasitic origin of the disease antiseptic or antiparasitic agents are mainly employed. The first step should be the removal of the scales, films, crusts, or sebaceous masses on the scalp. This is best accomplished by thorough washing and firm scrubbing of the scalp, using borax as a soap or a spirituous solution of *sapo viridis*. Afterwards precipitated sulphur, mixed with twenty times its weight of a basis of equal parts of lanoline and vaseline, should be well rubbed into the scalp at night and washed off in the morning with a liberal supply of pure olive oil. Mild cases of infantile seborrhœa yield to simple oily applications. In severe cases, salicylic acid alone, or in combination with the above-mentioned pomade of sulphur, is necessary in adults. When there is much excess of the oily secretion the sulphur may be advantageously employed as a 10 per cent. lotion in water to which a little alcohol is added.

SEPTICÆMIA

This subject is dealt with upon page 1473, under its allied condition, 'Pyæmia.'

SEROUS HÆMORRHAGE

This is a condition caused by a lowered coagulability of the blood, which manifests itself by the presence of œdema, urticarial eruptions, chilblains, headache, and slight albuminuria.

Pathology.—The condition was first recognised and investigated by Sir A. Wright, who gave to it the above title, after demonstrating that the underlying factor or pathological basis was a deficiency of lime-salts in the blood, which reduced the coagulability of the circulating fluid so that serous transudation into the lymph-spaces occurred.

The normal coagulation-time of healthy blood drawn from a finger-prick or needle-puncture of the lobule of the ear is about seventy-five seconds, when the experiment is conducted at an incubator-temperature of that of the body.

In the condition under consideration the coagulation-time may be lengthened to two minutes and thirty seconds or more, but after a few doses of chloride of calcium the period may be found to be shortened to the normal, and by continuing the administration all the symptoms and signs of the affection pass off.

Symptoms.—In addition to the presence of chilblains, urticaria or wheals, physiological albuminuria, and œdema

about the eyes, feet, ankles, and legs, G. W. Ross has shown that the condition often manifests itself in a severe, chronic, and hitherto intractable type of headache. This form of cephalalgia is usually present on waking as a dull, heavy aching experienced over the frontal or temporal region for several hours, which tends to diminish or entirely to pass off towards the afternoon. It occurs in individuals of the so-called lymphatic diathesis, hence it is styled by Ross 'the lymphatic type of headache'; and this observer has found that anorexia, constipation, anæmia, pain after food, cough, shortness of breath, and palpitation accompany the classic symptoms observed by Wright.

The pain after eating is regarded by Ross as due to the blood-condition, which, owing to the diminished coagulability, permits of serous hæmorrhage in the gastric mucosa when the membrane receives its full supply of blood as digestion commences. The transudation of the blood-plasma causes a rapid increase of pressure upon the nerve-filaments in the wall of the stomach, producing pain or a sense of fulness; and the last-mentioned observer points out that gastric crises are common in urticaria and in Werlhof's disease, and that they are relieved by the administration of lime-salts.

All the symptoms and signs of serous hæmorrhage may be made to reappear (after their dissipation by chloride of calcium) upon the administration of citric acid or citrate of potassium, which possesses the power of rapidly decalcifying the blood.

Treatment.—The condition yields rapidly to the administration of a calcium salt. The drug most frequently employed for this purpose is the chloride, given in doses of about 15 gr. three times a day in solution in chloroform water or camphor water. The lactate is much more pleasant to the taste and is equally efficacious; it may be given in similar dose and vehicle for two or three weeks at a time.

SHOCK AND COLLAPSE

The term 'shock' is usually applied to the state of profound prostration which follows injuries, accidental and surgical. It is, however, not of merely surgical interest, since an identical condition sometimes results from a violent emotion in which terror, pain, anger, or anguish has been predominant. In the great majority of cases of shock both factors have been in operation, the mental condition being associated with the traumatic.

The state is closely allied to that of *collapse*—a condition usually met with in cachectic conditions, generally the outcome

of toxæmia, as seen in cholera, diarrhœa, and the peritonitis following soon after severe abdominal operations, and sometimes observed in acute poisoning by various substances, organic and mineral.

Syncope is to be differentiated from both these conditions, since in typical shock and collapse the mental functions are not abolished, though depressed; and in syncope the state is usually of brief duration.

Pathology.—All three states are dependent, according to the researches of Mummery, upon a considerable fall in the arterial blood-pressure. In syncope this effects a cerebral anæmia which suspends the cerebral functions. In collapse the general blood-pressure falls through changes in the circulating fluid or from *inhibition* of the vaso-motor centres; whilst, according to the authority just named, surgical shock is the result of a general vaso-motor-paralysis, the direct outcome of *exhaustion* of the vaso-motor centres.

The risk of shock is in proportion to the extent of tissue-injury, being great in crushing accidents, especially when a large area of skin is involved along with nerve-trunks, and in abdominal operations where an extensive area of the peritoneum is exposed to manipulation in elderly subjects.

Symptoms.—In shock these follow very soon after the exciting cause which has exhausted the vaso-motor centres, whether this be psychic or traumatic. In collapse the profound prostration comes on more gradually, but ultimately the clinical picture, when fully developed, is almost identical in both conditions. The mental state is often one of apathy, though consciousness is retained. The physiognomy is characteristic: the face is pinched and drawn, the nose sharp, the eyes sunken, the lips cyanosed, and the pallid features are cold and covered by a clammy sweat. The extremities are also cold to the touch, and the general cutaneous surface may resemble that of the cadaver, though the skin is moist; the breath also gives no sensation of heat when the observer's hand is held in front of the mouth. The temperature in the axilla is far below the normal, and often this is the case also in the rectum; but where the collapse has supervened upon some pyrexial condition a high rectal temperature may be registered. The pulse is small, rapid, and irregular, and finally ceases to be felt in the radials, though the mind remains clear. The breathing is quick, sighing, or irregular, and the voice feeble.

The urine is often suppressed, especially when previous vomiting has been copious or where diarrhœa has existed; it is always scanty and liable to be retained in the bladder. Thirst is unquenchable and the tongue cannot be kept moist. In this condition of extreme prostration there is often no sensation of pain, as seen sometimes in perforation of the

stomach or after extensive burns or scalds, and the patient sinks with his faculties often clear to the end.

The sphygmometer from the first indicates a marked fall in the blood-pressure, and this is almost invariably associated with acceleration of the pulse, which latter sign Mummery states is roughly in proportion to the amount of shock.

Deferred shock is often witnessed after accidents in which the mental excitement seems to postpone for a time the supervention of the collapse; and this is not rare after serious wounds received in warfare. It is frequently observed also in grave traumata received during alcoholic intoxication. The phenomenon is also observed in children and neurotic subjects after operations which are followed by considerable pain.

Prognosis.—This is always grave. In the main the gravity of the case may be roughly estimated by the nature of the exciting cause or the duration of its operation: thus it is obviously very bad when it follows very prolonged abdominal operations. The first signs of recovery are evidenced by the return of warmth to the extremities, and often by the return of the original pain; the pulse returns to the wrist, and the respirations become slower and fuller.

Treatment.—In shock following operation preventive measures should not be lost sight of. The view is largely held that ether-anæsthesia is less liable to be followed by shock in protracted operations than when chloroform is used, since in the latter the lowering of the blood-pressure, according to Priestley Leech, is a constant feature during the entire period of anæsthesia. The C.E. mixture should always be preferred. Rapidity of operation is also a matter of considerable importance, and the avoidance of exposure to cold during the operation is a vital matter in protracted cases. The injection of cocaine into the main nerves of the limb about to be amputated diminishes the risk of shock and enables the operation to be performed without waiting for the initial symptoms of shock to pass off in severe accident-cases.

Warmth should be applied by various means to the body whilst the patient is kept in complete mental and physical repose, while the foot of the bed is raised and the limbs bandaged so as to retain as much blood as possible where it is most urgently required.

Morphia hypodermically is one of the most valuable aids in the treatment of surgical shock, and it should be immediately followed by large injections of hot normal saline solution. When urgency is extreme these should be given by a large vein, and a full dose of adrenalin injected with the fluid; in less pressing cases the saline may be thrown into the rectum or injected into the loose areolar tissue of the arm-pit or mammary region.

A most vital point insisted upon by Mummery is the avoidance of all stimulation. Since the shock is directly due to the exhaustion of the vaso-motor centres, stimulants like alcohol, ether, digitalis, and strychnine are not only useless, but fraught with grave danger. Ergot and adrenalin are not open to this serious objection, but they should be administered by the veins.

Collapse should be treated by free stimulation and the administration of copious liquids in those cases where the blood has been drained by exhaustive diarrhoea; in urgent cases saline injections are as clearly indicated as in shock.

SLEEPING SICKNESS, or TRYPANOSOMIASIS

This, also known as Negro or West African Lethargy, is a chronic fever produced by the presence in the blood of *trypanosoma gambiense*, and characterised by progressive lassitude, general lymphatic-gland enlargements, profound muscular feebleness, and wasting, terminating in death after a prolonged period of mental lethargy.

Distribution.—The disease has been long endemic in the tropical regions on the West Coast of Africa between the Senegal and Loanda rivers, extending inland as far as Lake Tanganyika, along the Congo. Of late years it has appeared in epidemic form in the Uganda Protectorate. Owing to the long incubation-period, the disease has been met with in the West Indies and in South America amongst imported natives.

Etiology and Pathology.—The native races suffer almost exclusively, but of late years Europeans have been known in several instances to succumb to the disease. The specific parasite is a protozoon known as *trypanosoma gambiense*. Thomas and Brein maintain that all the forms of sleeping sickness occurring in the different regions in which the disease is endemic or epidemic are produced by this organism. It is an actively moving worm-like body, one end being bluntly conical and the other terminating in a flagellum, the total length varying from 18 μ to 25 μ . A highly refractile spot or vacuole, as described by Dutton, is visible in the posterior part of its short thick body whilst a transparent undulating membrane projects along one side of the parasite. The flagellum stains crimson by MacConkey's method of employing Romanowsky's chromatic plan of staining. As the result of the experiments of Greig and Gray and others, there is no longer any doubt that this organism is the cause of the fever, lethargy, and other symptoms of the disease. It is found in

the blood and, in the great majority of instances, in the lymphatic glands, by a simple puncture made during life. In the later or sleeping stage of the disease it is found very generally in the fluid removed by lumbar puncture; but Christy, as the result of over a hundred lumbar punctures, concluded that the parasites in many instances never enter the cerebro-spinal fluid, though they may be seen in the blood in enormous numbers, and in both liquids they appear to come and go in a remarkable manner. Their appearance in the cerebro-spinal fluid at an early stage is likely to be associated with active brain-symptoms, as mania, &c.

The parasite is transmitted to man by the bite of the tsetse fly, *glossina palpalis*, and, though D'Aguiar states that the distribution of sleeping sickness and of this glossina are identical, it is held that other species may convey the disease, and glossina palpalis may introduce other trypanosomes which are not capable of producing sleeping sickness. Gray and Tulloch believe that there is no developmental cycle of the parasite in the fly, as in the case of the mosquito and the malarial parasite. Koch identified male and female trypanosomes in the fly, and was able to differentiate in this manner *gambiense* from other parasites of the same species, the developmental cycle occurring in the stomach of the insect, the males being slender and the females plump. He has also discovered that the fly is infected by sucking the blood of the crocodile which acts as the host of the parasite. This fact may explain why the fly keeps so close to river banks.

Pathological Anatomy.—The anatomical changes consist of enlargement of the lymphatic glands throughout the body, a small-celled infiltration around the vessels of the brain and spinal cord, and increase of the sub-arachnoid fluid, with dilatation of the cerebral ventricles and some flattening of the cerebral convolutions. All these changes have been produced in animals by the inoculation of the organism which causes the disease in man. Sometimes degenerative changes are found in the bone-marrow and hæmorrhagic foci in the spleen and stomach. Secondary changes are also common, as pneumonia, purulent meningitis, tuberculosis, pleuritis, pericarditis, dysentery, &c.

Symptoms.—These come on after a very variable and often prolonged period of incubation, which has been proven to extend in some cases beyond three or four years; and a longer period still is believed in by many observers. During this period the trypanosomes are in the blood or glands and no symptoms are produced; some investigators have reported the discovery of the parasites in the blood or glands in subjects who never showed symptoms of the disease. The long duration of the incubation-period, for instance, if this can be as long as seven years, as many believe, may account for

such a result; and, secondly, the trypanosome-organism detected may have belonged to a harmless species.

The first symptoms may be a general lymphatic enlargement, with some swelling of the parotids, without any fever, listlessness, or feeling of ill-health. This is followed, after a variable period, by smart fever, headache, muscular weakness, as shown by tiredness or exhaustion, and a similar mental feebleness or slowness manifests itself often with difficulty of speech, which is slow, thick, and indistinct; there are general tremors and the gait is staggering. The expression becomes dull, listless, markedly stupid or vacant, though the patient, after an interval, will answer any question put to him, but in as few words as possible. He will not eat unless aroused, and may fall asleep during his meal, and as the drowsiness progresses he can only be awakened with difficulty, and soon relapses into sleep or apparent stupidity; marked wasting supervenes. Finally the sleepy state passes into prolonged lethargy or coma, in which death occurs usually from some secondary infection or complication, as purulent meningitis, pneumonia, &c.

The duration from the first signs of infection by the parasite till death varies from two to six months, and is often longer when the patient is carefully fed.

Of the types of the disease in the Congo Free State, Dutton, Todd, and Christy have described several in which sleep is never a pronounced feature, death occurring apparently before the lethargic state has developed, and sometimes without showing evidence of the implication of the lymphatic glands. In the type of the disease met with in Uganda, lethargy is usually the predominant clinical feature. Death is the almost invariable rule once the unmistakable symptoms have developed; but the above-mentioned authorities describe a type of the disease met with in the Congo in which only lymphatic enlargement exists and no deterioration of health follows.

Convulsions, general oedema, maniacal symptoms, and severe pruritus have been frequently observed.

Treatment.—The only drugs known to possess any influence over the progress of the disease are atoxyl and trypan red or trypanroth, which is an aniline dye recommended by Ehrlich. This latter, in doses of 5 gr., should be given in conjunction with the atoxyl, which is an arsenical aniline salt for hypodermic use. One Pravas syringeful (1 c.c.) of a 20 per cent. solution should be injected daily till mild symptoms of arsenical poisoning begin to show themselves, after which the drug must be continued for long periods in reduced doses.

As a prophylactic measure, Koch proposes a vigorous crusade for the extermination of the crocodile in whose blood the causal trypanosome has been recently discovered.

SMALL-POX, or VARIOLA

This is an acute and highly contagious fever characterised by the appearance of an eruption of macules upon the third day following the onset; these pass through the stages of papulation, vesiculation, pustulation, and desiccation, often leaving permanent scarring.

The disease has been known from an early period in history, having been prevalent in China and India long before the Christian era. Since its first introduction to Europe 1500 years ago it has appeared in frequently recurring epidemics, often destroying millions of lives until the introduction by the immortal Jenner of vaccination in 1796, though Jesty, a farmer in Gloucestershire, had successfully vaccinated his wife and children with cow-pox in 1774. Improved sanitation and energetic preventive measures will probably prevent the disease ever being seen on the scale of any of the great epidemics of the past, except in uncivilised communities.

Etiology.—The disease flourishes in all habitable climates and regions throughout the globe, the aboriginal coloured races suffering most severely; but no race or age is exempt. One attack as a rule affords immunity, but second attacks are occasionally met with.

The disease is spread by contagion, and there can be little doubt that the germs are conveyed by inhaling the emanations from the body or lungs of a small-pox patient; they linger for long periods about the clothing and about the bedding, and may tenaciously persist for many months in infected houses and localities. Stokes maintains that the early skin-lesions prove that the virus in the first instance reaches the lungs by inhalation, and afterwards finding its way into the circulation, it selects the skin. The epithelial scales separating from the skin of a small-pox patient contain the germs in a highly active state, and these are readily conveyed to others by the clothes of nurses, of attendants, and of all individuals having access to the sick-room or to the convalescing patient.

The question of *aërial convection* has of late years attracted much attention. The opponents of this view maintain that the cases which spring up in the vicinity of small-pox hospitals can be easily accounted for by personal communication. The dissemination of the disease from the small-pox ships of the Metropolitan Asylums Board to the shore of the Thames at Purfleet appears to settle the question, since Thresh, who investigated the outbreak, came to the conclusion that personal communication played no rôle in the Purfleet outbreak of 1901, and that in favourable conditions of wind the influence of the small-pox ships extended for a distance of almost

three miles. Blyth stated that the influence of the Sheffield small-pox hospital could be distinctly traced for a circle of 4,000 feet. The disease is infectious from a very early stage, but the period of its most intense contagiousness is believed to be that of the desiccation-stage, and there are numerous instances of the infection being caught from the dead bodies of small-pox victims. Probably flies and other insects act also as carriers of the contagion. The old practice of inoculation proved that small-pox was easily conveyed by inoculating the contents of the small-pox pustule or vesicle; the disease so produced ran a mild course, but the individuals suffering from this modified form of the affection became foci for its spread, and healthy susceptible subjects received the disease from the inoculated patients in a highly virulent and often fatal form; hence the practice introduced by Lady Wortley Montagu into England in 1721 was pronounced illegal by an Act of Parliament in 1840.

Individual insusceptibility is rarer in small-pox than in any other contagious disease, and still a mild or modified type of the disease is very occasionally met with in individuals who have never been vaccinated, as emphasised by Warner of Leicester. There are also remarkable differences in the intensity and death-rate of the disease in different epidemics—a fact which is becoming less obvious since the universal practice of vaccination.

There cannot be a doubt about the parasitic nature of the disease, but the bacteriology of small-pox still awaits demonstration. De Korté has recently found certain bodies which he maintains are protozoan parasites in the vesicles, and from these amœbæ, spores of an active mobile character are discharged into the fluid of the vesicle. Love describes similar mobile bodies as having been discovered in the blood of patients suffering from the other exanthemata and from syphilis; but Porter states that he found these bodies in the blood of healthy subjects. Ewing holds that the amœba-like protozoan bodies discovered by Guarnieri and named by him *cytocyctes variolæ* are not parasites, but are the result of a peculiar degeneration of the protoplasm of the epithelial cells, though Guarnieri's researches were corroborated by Councilman, Calkins, and several others who traced their cyclic developmental changes.

Morbid Anatomy.—The changes are mainly confined to the skin and mucous membranes. In the skin the process commences in the upper and middle portions of the rete mucosum layer, the cells of which early become swollen and opaque; exudation of fluid occurs, with the formation of a reticulum, and the horny layer of the epidermis is raised, producing a clear vesicle. Owing to the degeneration of the proliferated cells the contents of the vesicle become purulent,

and umbilication or a central depression is observed. This latter phenomenon was formerly believed to be the result of the presence of a duct of a sweat-gland or hair-follicle, which prevented the elevation of the centre of the dome of the pustule; but it may be caused by any of the bands which are formed by the stretching of the rete-cells, and which give a characteristic loculated structure to the vesicle or pustule. It disappears at a later stage, when the pustule uniformly becomes distended with leucocytes. More or less evidence of active inflammatory changes is also observed in the papillary layer, and in the hæmorrhagic cases extravasations or petechiæ are found in the skin or in the pustules.

The pustules are found in the mucous surface lining the mouth, pharynx, and upper part of the œsophagus, and occasionally in the stomach and intestines. The heart shows marked myocardial changes, usually in the form of cloudy swelling, and occasionally acute fatty degeneration is witnessed. The spleen, kidney, liver, testicle, and bone-marrow show corresponding changes. Hæmorrhages may be found in all the viscera, between or into the muscles, and extravasations into the skin, into the mucous, and sometimes into the serous membranes are observed in the hæmorrhagic cases. Numerous secondary lesions, the result, according to Stokes, of infection by streptococcus pyogenes, may be found in the lung or other viscera in the form of local necrosis and thrombosis. The writer has witnessed a general phlebitis. The interior of the larynx may present the appearance of a sloughing diphtheritic membrane, which may extend down the trachea, the underlying mucous surface being sometimes extensively œdematous.

Symptoms.—The various stages of the disease are recognised as those of *incubation*, *invasion*, *eruption*, *secondary fever* or *maturation*, *desiccation*, and *desquamation*.

Stage of Incubation.—Though this period may be as short as eight and as long as fifteen days, it is remarkable how frequently it may be found to be exactly *twelve* clear days from the exposure to contagion till the initial rigor. During this stage there are no symptoms; usually the individual suffers from no feeling of malaise till just before the next stage.

Invasion.—This sets in as a rule suddenly or abruptly with a smart rigor or a succession of distinct shivering fits; in children a convulsion usually takes the place of the initial rigor. Severe lumbar and sacral pains are so constantly complained of that their intensity is a diagnostic point of considerable value at this early stage of invasion. Sir John Moore attributes this symptom to hyperæmia of the spinal cord, since it is almost always relieved by natural or induced bleedings. It may be intense in a case whose after-progress

shows the attack to be a very mild one. The backache is accompanied by severe frontal cephalalgia, often by vomiting, and not rarely by transient delirium and slight paresis of the lower extremities. In children, diarrhoea and stupor sometimes follow the initial convulsion. The temperature rises rapidly and may reach 104° by the evening of the first day, even in cases which afterwards prove to be mild; it usually reaches its maximum just before the rash appears, later on, when it may register almost 106° in exceptional cases.

With this prodromal or *primary* fever the usual accompaniments of restlessness, quick full pulse, thirst, anorexia, great prostration, and muscular or joint pains are observed; the skin may be hot and dry, but often there are profuse sweatings.

Prodromal or *initial rashes* are often observed at this stage before the true eruption of small-pox makes its appearance. These early eruptions are of great importance, and their liability to occur must be kept in remembrance in order to avoid serious errors in diagnosis; but, as regards prognosis they are of little or no consequence, since they may appear in the mildest as well as in the most severe forms of the disease.

Several distinct types of prodromal rash are recognised. Perhaps the most important of them is the *scarlatiniform* eruption, which appears as a bright punctate rash, sometimes upon the evening of the first, more frequently upon the second, day of the invasion. Like the other prodromal rashes of small-pox, this is best marked upon the lower portions of the trunk, extending over the groins and down Scarpa's triangle; it may be well out upon the flexor surface of the limbs whilst the extensors, face, and neck are quite free. This is usually classed as an erythematous eruption, but it may so closely resemble the rash of scarlatina that a diagnosis is only possible by realising that the throat and other signs of the disease are entirely absent. A second form of eruption consists of this punctate rash *plus* numerous petechiæ, which also, like the rash, spare the extensor surfaces. A third form is not rare in hæmorrhagic small-pox, in which, as pointed out by Goodall, the erythematous redness is very vivid and diffuse and accompanied by considerable swelling, so that erysipelas is simulated, especially as the face and limbs are affected. This form is also associated with petechiæ in the abdomino-crural and in the brachial triangles, and is soon followed by purpuric blotches, caused by larger extravasations into or under the skin. A fourth well-marked form of prodromal erythematous rash is the *measly eruption*, which, according to MacCombie, reaches its maximum within twenty-four hours after its first appearance, invading the entire cutaneous surface. It is, according to the description of this

observer, slightly, if at all, raised, disappears upon stretching the skin, and fades quickly. These latter features, with the absence of catarrhal symptoms and of Filatow's spots, differentiate the condition from measles.

Both these erythematous and hæmorrhagic types of prodromal eruption are very rare in children; they appear about the second day, and as a rule fade completely before the appearance of the true small-pox rash or very soon after, though the petechial spots may remain for a day or two as brownish stains. In severe petechial small-pox or purpura variolosa the hæmorrhagic rash may be combined with the scarlatiniform and may be a prodromal eruption, since it sometimes appears as early as the second day, and death may supervene before the true papular eruption has time to develop.

Eruption.—The specific eruption of small-pox appears upon the *third or upon the fourth day*. In *discrete* cases the period of invasion usually terminates by the end of the third day—viz. the eruption appears on the fourth. In the *confluent* cases the period is usually twenty-four hours shorter, the eruption appearing upon the third or, in very severe cases, upon the second day. It must be always remembered that though the shorter the period of invasion the greater is the danger of the case turning out to be of the confluent type, nevertheless there is, as emphasised by Sir John Moore, the notable exception of severe hæmorrhagic or purpuric small-pox, in which the specific exanthem may be postponed for several days or almost for an indefinite period.

The rash makes its appearance first upon the forehead, face, and scalp, the flexor surface of the wrists, then upon the neck, trunk, and lower extremities. At first, if carefully watched for, the exanthem is seen to be composed of minute *macules* or hyperæmic specks, which are level with the surface and disappear upon pressure. By the end of twenty-four hours the macule, which was somewhat smaller than a flea-bite (*i.e.* about $\frac{1}{10}$ in.), has become transformed into a raised, hard, conical *papule*, feeling to the touch like a particle of shot placed under the skin. This stage of the eruption reaches its full development by the third day—*i.e.* upon about the sixth from the initial rigor. The papule a little later becomes a *vesicle*, and by the termination of the fourth or fifth day after the first appearance of the macule it is visible as a hemispherical bladder of clear or slightly opaline fluid. Towards the end of this stage of the eruption (by about the seventh or eighth day of the disease), when the opaline vesicle has reached its final development and is about to become a *pustule*, the phenomenon already referred to as *umbilication* is observable. Its centre is depressed or tied down and the semi-purulent contents are distinctly loculated, so that if the vesicle be punctured by a fine needle, like the vaccinc-

vesicle it is found not to collapse, only a small portion of the milky fluid escaping through the puncture.

By the end of the ninth or upon the tenth day of the disease in a discrete case the eruption has reached the stage of its fullest development or maturation, each vesicle having now become completely transformed into a hemispherical pustule or small abscess, the umbilication having disappeared and the loculi destroyed, so that a needle-puncture causes complete discharge of its purulent contents. There is considerable pain, tension, and local swelling between the pustules, especially in regions where the skin is lax, as about the eyelids, which are often entirely closed. Each discrete pustule is surrounded by a zone of hyperæmic redness as the maturation, commencing upon the face, extends to the trunk and limbs.

Crusts or scabs form as desiccation proceeds, the purulent contents drying up sometimes after the pustule has exuded an adhesive amber-coloured liquid, and the crusts resulting from the hardening of this begin to fall off from the face by about the end of a fortnight, the process being delayed upon the trunk and extremities till several days later. Upon the trunk and limbs, owing to the pressure or friction of the bed-clothes, the pustules are often ruptured; and this constantly occurs in the confluent type, so that a foul-smelling discharge saturates the patient's bed-linen and cakes upon his body.

Next to the face, scalp, and wrists, the eruption is generally more profuse upon the back and upper portion of the chest; the regions invaded by the prodromal rash—*i.e.* the abdomino-crural triangles—often escape with a few isolated pustules. The rash invades the visible mucous membranes, the conjunctivæ, nasal lining, pharynx, and buccal cavity; and Moore's drawings show the entire digestive and respiratory tracts studded over with the typical pustules.

Desquamation.—This stage follows the falling of the crusts. As these drop off the skin underneath them is shed in successive small flakes, often leaving permanent scars or pitting; but usually little or no visible deformity results, except in the confluent type of the disease.

Fever.—To return to the constitutional symptoms present during the eruption. The initial high temperature or *primary fever* falls upon the appearance of the true small-pox exanthem, and in mild cases the thermometer registers the normal degree of temperature, the fall being either sudden or extending over twenty-four hours. At this crucial period in the diagnosis the papular or vesicular eruption is usually well developed, though the patient may express himself as feeling quite well, having lost all the severe backache and cephalalgia, and is able to walk about; this, combined with the normal state of the body-temperature, is very liable to lead to a serious error in diagnosis, unless it be remembered that such

is the almost pathognomonic feature of small-pox at this stage of its progress. No other exanthematous affection presents such a well-defined thermometric feature.

In confluent cases the temperature also drops, but the relapsing type of pyrexia is most frequently met with, the primary fever falling suddenly to the sub-febrile range in the morning and rising towards evening to 102° or 103° , or else the drop is a slow one, the normal line being scarcely reached.

With the development of pus in the vesicles the *secondary fever* or fever of maturation appears, usually about the eighth or ninth day after the initial rigor. It is generally less severe and lasts but a few days in mild discrete cases, in which sometimes it is entirely absent; but in confluent small-pox its duration is indefinite, running a high course, with morning remissions, and in severe examples of this type it may be either continuous or markedly remittent or irregular, with exacerbations which reach to hyperpyrexia as the fatal issue approaches. The secondary fever is evidently a septic phenomenon resulting from the absorption of some element of the contents of the pustules. With the progress of the fever at this stage the usual accompaniments of high temperature are present, as thirst, anorexia, profuse sweatings, and sometimes chills; the pulse, which had become normal, quickens, delirium is not uncommon, and a vicarious diarrhœa may set in.

As recovery is approached the temperature falls slowly, with slight irregularities in the curve of the protracted lysis.

VARIETIES OF TYPE

Several distinct forms of the disease are recognised, but these sometimes run into each other, so that any elaborate classification may appear more or less artificial.

There are (1) *Natural small-pox*—this is the disease unmodified or altered by vaccination; (2) *Varioloid* or *modified small-pox*, in which previous vaccination by cow-pox mitigates the characters of the affection. (1) Of the natural small-pox, or *variola vera*, there are three well-marked varieties: (a) *Discrete*, (b) *Confluent*, (c) *Malignant*. The last group (*malignant*) corresponds to or is synonymous with *variola hæmorrhagica*, and includes two different varieties of the disease—viz. *purpura variolosa* or *black small-pox*, and *variola hæmorrhagica pustulosa*.

Discrete, sometimes also called *distinct*, *small-pox* is the affection, already described in the preceding pages, in which the characteristic eruption of macules, papules, vesicles, pustules, and scabs or crusts appears sparsely scattered over the cutaneous surface and widely separated from each other by regions of healthy or unaffected skin. The rash appears, as stated already, usually upon the fourth day, and in mild

cases pustulation may be scarcely, if at all, recognisable, so that, though the primary fever and initial symptoms may be smart and even severe, there is sometimes no secondary fever at all. The disease very rarely ends in death—never, indeed, unless some complication occurs; but it may give rise to the most virulent type in healthy individuals catching the contagion from a patient who only exhibits a few vesicles upon the face or wrists.

Confluent Small-pox.—In this variety the distinguishing point is the extent and thick-set nature of the eruption, which may cover the entire body and may appear upon the second or upon the third day; rarely is it delayed to the fourth. The initial symptoms are usually more severe than in the discrete form. Even in severe cases the rash may be confluent on the face and hands—that is, the papules may be so thickly studded over the surface as to leave practically no healthy skin between them, whilst it remains discrete over the trunk. In the early papular stage, however, though the papules are very thickly set and there is much swelling, it is often only when vesiculation or pustulation is established that the eruption appears really as one continuous sheet or mask, causing the hideous deformity of the swollen face and eyelids which is so characteristic of the disease. With the constitutional symptoms common in the discrete form there are found others added on, which have grave diagnostic significance—viz. diarrhœa of a severe type and profuse salivation, with swelling of the tongue; delirium is common even at an early stage, and the visible mucous membranes are invaded by the eruption. The hoarseness, pain in swallowing, vomiting, coughing, deafness, and purging may be interpreted as the results of the early involvement of the mucous membranes which are beyond the reach of sight.

Though the temperature falls upon the appearance of the eruption, as in the discrete form, the normal is seldom reached even when all the other symptoms are markedly ameliorated; the swelling of face, hands, and feet, though not so severe as at a later stage, is nevertheless well marked.

The secondary fever usually appears about the eighth day after the invasion; this is severe and irregular, but sometimes continuous, with very sharp exacerbations. A day or two later the maturation of the vesicles has converted the face, hands, and feet into one continuous sheet of suppuration in each region, the swelling or œdema and hyperæmia being excessive. Delirium, if absent up to this point, now becomes marked; the pulse quickens greatly, thirst is intense, and diarrhœa may return in a very persistent form. The lymphatic glands swell and symptoms of œdema of the glottis or any of the numerous pulmonary, renal, ear, eye, circulatory, or other complications, may supervene. Abscesses, gangrene,

bed-sores, and erysipelatous inflammation of the skin are common in severe cases. Death is most frequent about the twelfth day, but may occur at any period later on during the convalescent stage.

In the cases which end in recovery the desiccation-stage is prolonged, and as the large crusts or plaques separate from the skin ulcerated spots of various dimensions are visible; these become covered over again by successive scabs or crusts, which are periodically shed. In this way the secondary fever, which usually fades or disappears by the middle of the third week, may be protracted into the fourth or even fifth week. The healing of the ulcers by scar-tissue which undergoes the usual cicatricial changes leads to the characteristic 'pitting' which permanently deforms the face. This result, so commonly observed in former years before the practice of vaccination, is now comparatively rarely witnessed. The hair is always shed and the nails sometimes drop off, but permanent baldness does not usually follow.

The name *semi-confluent small-pox* is sometimes applied to the above condition, in which usually the eruption is confluent upon the face and hands and discrete upon the trunk. It should, however, be restricted to those cases where the pustules, though thick-set and close to each other, do not coalesce, since the term implies a lesser degree of severity; whilst it is a well-recognised fact that the gravity of the disease is in proportion to the confluence of the eruption upon the face, even when the pustules are sparse and discrete upon the trunk.

Corymbose small-pox is a rare form of the disease in which the pustules coalesce in clusters chiefly in the neighbourhood of the arm-pits, groins, or backs of the knees, and it is of very grave import.

Malignant or *hæmorrhagic small-pox*, in the typical purpuric form known as *purpura variolosa* or black small-pox, is almost invariably fatal. The disease is hæmorrhagic from the start, the eruption of purpuric spots appearing on the second or third day in combination with a diffused or punctate erythematous redness, which generally follows symptoms of an unusually severe invasion. The purpuric spots show as true petechiæ, vibices, or small ecchymoses, which rapidly enlarge, sometimes producing an almost universal purple lividity approaching to blackness. Hæmorrhagic extravasations appear upon the swollen face and conjunctivæ and upon the visible mucous membranes, and extensive bleedings occur from the gums, mouth, nose, bronchial surface, stomach, kidneys, bowels, and, in women, sometimes from the uterus; the pulse and respiration rate are greatly accelerated. Death usually occurs before the appearance of the true exanthem of the disease about the fourth day, with the patient's mind

remaining clear to the end. Sometimes, but rarely, the purpuric spots brighten up, and a delayed abortive type of rash appears before death.

Sir John Moore describes a form of hæmorrhagic small-pox, also without the formation of the characteristic exanthem, in which the purpuric condition reaches its extreme limit in an acute hæmophilia, the patient bleeding from every pore or orifice of the body, and even weeping tears of blood. It differs solely from the purpuric in its terrible intensity.

Variola hæmorrhagica pustulosa is the form in which small-pox progresses as in the severe discrete or confluent type up to the last stage of papulation or of vesiculation or to the beginning of pustulation, when suddenly numerous hæmorrhagic extravasations occur into the halos around the vesicles or pustules, and these latter also fill up with blood. There are bleedings also from the mucous surfaces, as in the purpuric form, and death is the almost constant rule, its date being usually determined by the period at which the hæmorrhagic extravasations supervene upon the ordinary eruption, being earlier when the papules have become hæmorrhagic than when the bleeding occurs into the pustules. In rare cases, when the eruption remains discrete on the face recovery may ensue; the secondary fever in this form seldom has time for its full development, since the fatal event usually occurs before the ninth day.

Varioloid or *modified small-pox* is the type of the disease usually met with in individuals who have been thoroughly vaccinated. It differs in no way save in its mildness from the discrete form of true variola, and may be sometimes seen in those rare unvaccinated subjects who possess a strong natural insusceptibility to the disease, and also in those who have suffered from an attack of small-pox many years before. The invasion may, however, be as severe and the backache as excruciating as in a smart attack of natural small-pox. As a rule, the rash consists of a few papules confined to the face or wrists; these usually appear upon the fourth day of the invasion, but it is much more common to witness the presence of the erythematous prodromal eruptions (already described) in this form of the disease than in the natural small-pox. The advent of these heralds therefore, as a rule to which there are few exceptions, proclaim a mild or modified attack of the disease.

The few scattered discrete papules very generally abort—that is, they either fail to vesiculate or to pustulate; hence very usually there is no secondary fever at all, the papules or vesicles shrivelling up, so that the patient, after the abrupt drop in the initial fever, becomes practically convalescent, though he may disseminate the disease in a virulent form amongst others. In other cases the eruption, though sparse,

runs through the typical changes observed in the natural variety of the disease, but the number of pustules being so few the secondary rise of temperature is either insignificant or absent. When the development of the papule stops short at vesiculation it shrivels up and remains dry—a condition to which the terms *wart-pox* and *horn-pox* have been applied.

There is still another rare and interesting variety of modified small-pox in which, after the invasion-period, no eruption at all appears, though there may or may not have been a prodromal erythematous or roseolar rash. This form is named *variola sine variolis*, and the patient convalesces immediately after the abrupt fall of the primary or initial fever, which is accompanied by severe back- and headache and even by transient delirium and vomiting. The nature of this latter anomalous condition has been several times demonstrated unmistakably by a second person catching the disease, even in the confluent form, from the patient who never developed a single papule during his brief illness.

The amount of protection afforded by vaccination depends upon many circumstances, as the thoroughness of the operation, the time which elapses between its performance and the exposure to contagion, and possibly also upon individual peculiarities. These will be referred to later on; they afford an obvious explanation of the degree of mildness or of severity in which the modified disease appears in vaccinated subjects.

Complications of Small-pox.—These are numerous, and many of them are of a serious nature. The skin involved in the eruption may slough and extensive gangrene result, as seen in the scrotum, or large bed-sores may form. During convalescence successive crops of boils are common; these may be associated with deep-seated multiple abscesses, especially in the convalescence after confluent attacks. The condition was described by Trousseau under the title of the *acute furuncular diathesis*. Acne of the pustular type belongs to the same category, and erysipelas of phlegmonoid character is also occasionally met with.

The eyes suffer severely sometimes, conjunctivitis, keratitis, iritis, or retinitis resulting, and in serious cases a panophthalmitis or suppurative inflammation of the eyeball, the result of metastatic abscesses, eventuates in permanent blindness. Otitis media, which may be associated with bone-disease and sinus-thrombosis, is not a rare event. But perhaps the most frequently met with of the serious complications is bronchopneumonia, which is a common cause of death.

Pleuritis and pericarditis, when present, are liable to be of the suppurative type; both these are, however, rare, though often the heart-muscle shows evidence of serious impairment. Laryngeal complications are common, the aphonia or hoarseness which sometimes appears early in the disease when the

eruption invades the larynx is liable to end in a fatal œdema of the glottis. If this does not occur, at a later stage there is the further danger of a perichondritis setting in, which may eventuate in erosion of the glottis or cartilages, leading to aspiration-pneumonia, and eventually followed by permanent stenosis should the patient recover. The laryngeal inflammation is usually accompanied by a more or less severe tracheitis and bronchial catarrh, the latter being an almost constant condition in severe confluent cases. Diphtheria may complicate the mucous-membrane affection, as erysipelas sometimes follows on the cutaneous condition.

Renal complications show their presence by albuminuria and hæmaturia; the latter sign is the most frequent accompaniment of hæmorrhagic small-pox, but true nephritis, so common in scarlatina, is not often met with. Of the digestive disturbances, diarrhœa with melæna, and vomiting with hæmatemesis, may be distressing, and the dysphagia from the throat-complications or from the acute phlegmonous glossitis sometimes seriously affects alimentation.

As in mumps, when the parotid becomes inflamed in small-pox the testes and ovaries may also suffer; but in small-pox the parotitis is very prone to eventuate in suppuration.

The secondary sepsis may show itself by a suppurative arthritis, constituting a true pyæmic condition, and the bones not rarely suffer erosion or necrosis.

The implication of the nervous system may manifest itself by wild delirium or convulsions, which are really symptoms of all the severe confluent cases; but sometimes meningitis, sinus-thrombosis, or cerebral abscesses supervene, producing marked head-symptoms. Various forms of paralysis and aphasia may eventuate from these latter conditions, but more frequently the loss of motor power following small-pox is either spinal or of the multiple neuritis type, caused by the toxins of the small-pox organisms or by those of the concomitant diphtheritic complication.

Diagnosis.—This is of vital importance in the early stage, owing to the highly contagious nature of the disease demanding rigid isolation. The diagnosis of the various prodromal rashes has been already fully detailed. The presence of an epidemic of the disease should suggest the serious nature of all illnesses of a febrile type in which severe backache and headache are ushered in with shivering or vomiting. The presence or absence of vaccination-marks is of great importance; thus in the vaccinated the prodromal rashes occur most frequently, if not exclusively. The preventive value of vaccination is so unmistakable that a *recent* successful vaccination may be accepted as practically excluding small-pox.

Of the diseases likely to be mistaken for variola in the stage of invasion, *influenza* must not be forgotten. It affords

great, if not insurmountable, difficulty, as the backache and shivering may be as severe as in many cases of small-pox ; and, moreover, the writer has witnessed numerous examples of influenza appearing with a transient erythematous rash, accompanied by backache and *transient paresis of the lower extremities* exactly like those in typical small-pox—so that the diagnosis must be delayed till the third or fourth day ; and even then, should the fever drop abruptly, a close search should be made for any odd papules scattered throughout the hairy scalp, which might be the sole representatives of a variolous exanthem. The backache of *lumbago* should never be confounded with that of small-pox ; it is always apyrexial and often only experienced upon movement.

Typhus fever need hardly be thought of, unless a coincident epidemic be present. As pointed out by Goodall, the petechiæ of typhus may be mistaken for the rash of hæmorrhagic small-pox (*variola purpurea*) ; they are, however, preceded by the rose-coloured maculæ, which appear upon the fifth day of the invasion of typhus ; these latter at first disappear upon pressure, and are not accompanied by subcutaneous hæmorrhagic extravasations, as in *variola*. The characteristic drop to normal in the temperature of small-pox upon the first appearance of the eruption is almost pathognomonic of the disease, even should this appear to an inexperienced physician to be different from his previous conception of the small-pox exanthem. This fact helps to exclude *measles* early in the confluent type of the disease, the only form of small-pox likely to be mistaken for measles ; and, moreover, the shotty hard feel of the multiple and closely set papules of confluent small-pox is never found in measles. In measles the papules cannot be felt by the fingers if the skin over them is stretched (Grisolle's test) ; and catarrhal symptoms are always present. *Scarlatina* is only likely to be confounded with the prodromal rash ; the true variolous exanthem never simulates the rash of scarlatina.

Chicken-pox or *varicella* is usually easily differentiated by the predominance of the eruption on the back and front of the trunk ; by the short period of invasion, which hardly ever exceeds twenty hours, and is often so mild as not to be noticed ; and by the character of the eruption. This commences with papules, which in less than twelve hours are usually all converted into vesicles, some of which are oval ; whilst, according to MacCombie, an oval vesicle never is seen in small-pox. Umbilication is seldom observed in any of these vesicles, and their unilocular formation is further demonstrated by their complete collapse when punctured by a fine needle. They come out in successive crops, so that papules, plump vesicles, pustules, and even some in the stage of desiccation may be all visible at the same time, and they never coalesce or become

confluent. In adults, however, the difficulty of a correct diagnosis may sometimes be insuperable. Thus, in a case recently witnessed by the writer in a Brazilian gentleman, who a fortnight previously had been in a region probably infested by small-pox, the disease set in abruptly with a rigor, severe fever, backache, headache, and vomiting; there was a profuse vesicular eruption covering the face and entire body, including the hands, feet, and visible mucous membranes. The fever had not abated markedly, however, upon the advent of the eruption, as in some severe cases of confluent small-pox, and the appearance of grave illness together with many examples of distinct umbilication in the vesicles rendered the diagnosis of small-pox much more than probable. The patient was seen by Dr. Robb and promptly isolated in hospital. The fact of its being a case of true varicella was afterwards demonstrated by this latter disease appearing in its typical form in an individual brought into contact with the patient in the convalescing stage.

Purpura (either simplex, Henoch's, or Werlhof's) is differentiated from the purpuric variety of small-pox by the absence of the severe backache and of the formidable fever. The general gravity of the case from the onset should arouse suspicion in malignant small-pox even in the absence of any traces of a papular eruption. The ordinary hæmorrhagic form of small-pox cannot easily be mistaken for purpura, since the petechiæ in these cases supervene upon the papular, vesicular, or pustular condition of the eruption.

Certain skin-diseases may simulate discrete small-pox, when the patient first comes under observation in the apyrexial period, with a sparse eruption out on the face and hands. Especially in modified small-pox it may be difficult to exclude *impetigo contagiosa*; but a careful scrutiny of the history of the development of the eruption will clear the case up, and the absence of severe backache before the appearance of the pustules negatives small-pox. The bullæ of *pemphigus* are vesicular from their first appearance, and this is true of all the varieties of *herpes*; and, moreover, in both these affections there are no marked prodromal symptoms. *Acne*, *urticaria*, and most other conditions like *erythema multiforme* are excluded by this latter consideration alone.

Erysipelas of the face closely simulates a confluent eruption in this region, but an inspection of the entire cutaneous surface will invariably reveal discrete papules or vesicles upon the trunk or hands in this variety of small-pox.

Syphilis.—The papular or pustular eruption in this disease may be mistaken for small-pox in the presence of an epidemic; but the history of the case, if investigated properly, should never leave a doubt about the diagnosis; and other evidence of syphilis is invariably present.

Glanders affords a serious difficulty when it occurs in the acute form, with severe malaise, fever, and muscular pains followed by a papular eruption. The latter, though papular, seldom adheres to the uniform regularity in size of the small-pox papules; there are dense infiltrated areas or nodes in the skin, and these tend to rapidly break down and ulcerate. The papules in glanders speedily become pustules of larger size than those of variola, the vesiculation-stage being very brief. The pustules may be as large as a sixpence in diameter, and are hemispherical or have flattened tops, but rarely true umbilication is observed; they conform to the type of bullæ more closely than to that of a simple pustule, and they come out in successive crops. The history of the case, together with the evidence of the site of inoculation and the presence of a sanious foul nasal discharge, occurring generally in grooms or stable-boys, is almost pathognomonic.

Cerebro-spinal or so-called 'spotted fever' may closely simulate the purpuric variety of small-pox when the petechiæ are numerous and widely diffused; when herpes or ecthyma are also present the case may resemble the other hæmorrhagic type of variola. The retraction of the head and severe pain down the upper portion of the spine, the presence of Kernig's sign, with tremor and rigidity of the muscles, are, however, usually pathognomonic; but if doubt still remains, it will be set aside by lumbar puncture and the demonstration of the diplococcus intracellularis.

Leucocytosis is a constant phenomenon in unmodified small-pox, and may possibly prove useful in diagnosis; it is observed at the end of the first week and again in a more marked form about a week later.

Prognosis.—Even in the earliest stage of the invasion a fairly accurate forecast can often be made by an investigation into the vaccination-history of the patient. If this operation has been thoroughly performed and two or three well-marked scars are visible, the disease is almost certain to assume the modified type; the mildness of the symptoms is proportionate to the shortness of the time between the operation and the attack. Re-vaccinated persons seldom are attacked, unless a long interval exists between the second operation and the exposure. The mortality amongst unvaccinated patients of all ages amounts to nearly 50 per cent., and probably does not exceed 10 per cent. amongst those who have not been vaccinated since infancy, including those who only exhibit imperfectly formed and single scars. The all-round mortality amongst the re-vaccinated probably does not exceed 2 per cent. The diminution in the death-rate and in the incidence-rate of small-pox, due to vaccination, is more fully dealt with in the article upon 'Vaccinia.'

The death-rate in hæmorrhagic cases is practically 100 per cent., and more than 50 per cent. of the confluent cases die. In children under five years of age the mortality of all varieties of the disease is very much higher than 50 per cent.

Death, as already stated, may occur in the hæmorrhagic cases as early as the third day, rarely is it protracted beyond the sixth; whilst in the confluent type the fatal issue is usually to be expected about the twelfth or thirteenth day. Those who pass safely through the secondary fever may perish from any of the septic complications arising as late as the fifth or sixth week.

The prognosis, in the absence of complications, is fairly indicated by the profusion of the eruption upon the face and hands, and is always good when the pustules remain discrete; but the severity of the initial backache, headache, and pyrexia often bears no relation to the danger, being sometimes very severe in the modified form of the disease. It is usually, however, a grave omen when the fever does not drop completely after the appearance of the papules. In unvaccinated patients who escape with their lives the convalescence, as pointed out by Moore, is as a rule greatly protracted, and it need scarcely be emphasised that the percentage of those permanently deformed by serious pitting is very high; the modified disease leaves little trace of its previous existence in the face or elsewhere. Pregnancy seriously deepens the gravity of the outlook, abortion or premature labour usually setting in with its accompanying septic dangers or hæmorrhages.

Treatment.—*Preventive* measures are of first importance in all epidemics, and the most rigid isolation of the patient in a properly equipped fever-hospital is essential. The administrative details now carried out in every properly constituted municipality consist in the immediate destruction of all bedding, clothes, &c., of the patient as soon as prompt arrangements for his isolation are decided upon, and the thorough disinfection of the house in which the disease has occurred and the disinfection of the clothing of those who have been in contact with the patient. Every individual who has been known to be in contact with a case should be vaccinated, unless the operation has been already performed within the previous year or two. In order to carry out disinfection and re-vaccination satisfactorily the machinery of notification is put into motion and the movements of the patient and the source of his infection investigated.

It will be found necessary to make provision for the isolation of suspects; all children in the immediate vicinity of the infected house should be prohibited from school-attendance.

Strict attention to the disinfection of the convalescent before he is permitted to mingle with the healthy is of vital

importance, as the disease is easily spread by those who still possess crusts or scabs upon the skin ; and burial direct from the small-pox hospital must be insisted upon, since the emanations from the cadaver are highly contagious.

Vaccination of patients already suffering from the initial symptoms of small-pox is of no value either from a diagnostic or therapeutic point of view, since both diseases (vaccinia and small-pox) may run their normal course in the same patient at the same time. But vaccination may usually be relied upon as a preventive even after the healthy individual has been exposed to the disease, provided it be not delayed beyond a period of three days. Even if vaccination be delayed for an entire week after exposure, though the vaccinated individual catches the disease it is extremely probable that it will be in a modified form. These considerations, however, should never prevent vaccination or re-vaccination being performed at the earliest possible opportunity after the discovery of an individual's exposure to the contagion of small-pox.

Were periodical re-vaccination compulsory, it is highly probable that the disease would never be seen in vaccinated communities ; even as it is, with re-vaccination never resorted to except in the presence of a prevailing epidemic, the mortality of small-pox in England, according to Sir John Moore, has fallen in the decade before the last to one-seventieth part of the death-rate of pre-vaccination times.

The treatment of the disease, when once established, must be conducted upon the lines of other fevers. Serum-therapy, antiseptics, and all known drugs have proved futile in cutting short the progress of the disease. Symptoms may be alleviated by the judicious administration of medicine and other agents ; thus restlessness and insomnia can be relieved by sedatives, delirium by ice to the head, and high fever by sponging. Of the methods tried to prevent pitting little need be said ; most of them are founded upon fallacies. Stokes' principle of excluding the air, keeping the parts permanently in a moistened condition, and using such emollient applications as diminish the local irritation, is the best, and it may be carried out by a lint-mask saturated with solution of boric acid or other antiseptic poultice, or by smearing the face over with any mild antiseptic ointment. Finsen's red-light treatment has been proved to be of no avail either in lessening the degree of suppurative fever or in the prevention of pitting.

The graver symptoms of the confluent and hæmorrhagic types must be met by free stimulation and the administration of chloride of calcium, which by increasing the coagulability of the blood lessens the hæmorrhagic tendency ; moreover, the latter drug, as emphasised by Sir Lauder Brunton, exerts a highly beneficial tonic action upon the cardiac muscle.

SOFTENING OF THE BRAIN.

(See under CEREBRAL EMBOLISM, p. 168.)

SORE-THROAT. (See under PHARYNGITIS, p. 1287.)

SPASM AND TREMOR

The increased and involuntary muscular action eventuating in spasm is a symptom of many diseases. As already stated under 'Convulsions,' page 240, spasms are either *tonic* (or continuous), as in tetanus, tetany, strychnine-poisoning, cerebro-spinal meningitis, writer's cramp, hysteria, &c. or *clonic* (intermittent), as in epilepsy, puerperal eclampsia, uræmia, infantile and hysterical convulsions, &c.

The movements observed in Facial Spasm (described under its own heading, page 420) illustrate the chief characteristics of a purely spasmodic affection confined to a limited number of muscles; the spasms are clonic, and, like those of universal distribution, are often heralded by a tonic spasm of brief duration. When tonic spasm is persistent it is known as *rigidity*, and by the term *contracture* when the persistent rigidity confined to certain groups eventuates in permanent deformity through shortening of the muscles, which undergo structural changes.

Tonic spasm is usually spinal, though it may be caused by cerebellar or pontine diseases; it is due to direct stimulation or irritation of the corresponding nerve-tissue, but more frequently it is the result of an exaltation of the reflex centre through the withdrawal of an inhibitory influence. As the severity of the tonic spasm passes off, the tension of the muscles sets up a clonus, so that a condition of Brown-Séquard's spinal epilepsy is produced and a true clonic spasm succeeds, which can, however, be often arrested by a strong painful impression on the sensory nerves of the part.

Clonic spasms are commonly of cerebral origin, being produced most frequently in the cortex by organic lesions, as meningitis, softening, hæmorrhage, or tumours, in which case they have usually a local commencement and afterwards become general. As in idiopathic epilepsy, though the cortex is the probable origin, the lesion is supposed to be of the nature of an over-action of the cells of the grey matter, possibly combined with imperfect nutrition.

Habit-spasm is a condition usually commencing in late childhood, commonly in neurotic subjects. It usually takes the form of some grimace, as winking, blinking, mouth-twitching, elevation of the chin, head-nodding, &c., but it

may be confined to jerking of the arms or shuffling with the feet, not unlike what is seen in chorea; a most objectionable form is that in which sniffing or grunting occurs through the deranged innervation of the respiratory mechanism. This form of spasm may be permanent even when its origin can be traced to voluntary imitation. Occasionally it can be inhibited by a strong voluntary effort, but returns when the mind is directed to something else. Usually, as in chorea, the condition is aggravated by the consciousness that the individual is being watched.

The prognosis depends upon the duration of the spasm; under judicious moral treatment, combined with arsenic and bromides, the condition is often amenable if taken in its early stage.

Myoclonus Multiplex is a form of clonic spasm allied to that described under 'Electrical Choreia' on page 217, but the affection is not associated with any danger to life. It was first described by Friedreich, under the title of paramyoclonus multiplex, as a severe clonic spasm affecting the limbs of an adult. A brief description of its nature will be found upon page 1075.

Saltatoric Spasm is the name given to a rare variety of clonic spasm whose presence is only brought into evidence when the individual makes an attempt to stand upon his feet. This action produces a series of contractions of the leg and thigh flexors, each alternating with a similar activity in the extensors, so that a number of jumping movements are produced upon each attempt, which sometimes lift the patient's body from the ground. In the recumbent posture the state of the limbs is normal.

Myotonia Congenita, or *Thomsen's Disease*, is characterised by a peculiar transient tonic spasm, described upon page 1073, and upon page 1075 a similar form of spasm is referred to under the names of congenital or ataxic paramyotonia.

The spasmodic movements of chorea are described under the heading of this disease, the spasms being associated with incoördination and muscular weakness. Under 'Paralysis, Spastic,' a similar combination of muscular weakness with permanent tonic spasm is described, and it is a feature in all the secondary sclerosis of the cord occurring in early and late life.

TREMORS

When clonic spasms are confined to isolated muscular fibrillæ the phenomenon known as *fibrillary* twitching is observed. The origin of these partial contractions is generally a local one (confined to the muscle), since they are so readily produced by a slight tap or direct stimulant; but certainly fibrillary twitchings may be of centric origin, though

rarely. These fine clonic spasms are seen in bulbar paralysis, progressive muscular atrophy, and in cachectic states. The term *tremor* is applied to clonic spasms of a coarser type than fibrillary twitching; in this latter condition the contractions are so fine that they are not able to effect any movement, since the muscle as a whole is not thrown into action. In tremor there are alternating contractions and relaxations of opposing muscles, which enable the origin and insertion of the affected muscles to be brought nearer to each other, the range of motion being trifling in most instances.

The tremor of paralysis agitans is described under its own name. There are five to seven oscillations per second, and the tremblings of the affected muscles continue during rest, the tremor being said to be *spontaneous*, in contradistinction to the intention-tremors, which only appear upon some attempt to bring the muscles into action.

Senile Tremor also occurs during rest. It closely simulates the fine tremor of paralysis agitans, but the head is more constantly affected and there is no muscular weakness or rigidity. The disease first appears in the arms; as implied in its name, it is sometimes an accompaniment of old age, and appears occasionally to develop into the typical agitans type, but more frequently it remains like the *simple* tremor which occurs very occasionally in the hands, head, and face of younger subjects. This form is often under the control of the will for a short period; it is readily excited by movement, whilst it commonly ceases during rest, and it is exaggerated by emotion and excitement of all kinds.

The tremors associated with chronic alcoholism, hysteria, mercurial poisoning, rickets, general paralysis of the insane, hemiplegia, exophthalmic goitre, &c., are described under the heading of each disease.

The tremor of disseminated cerebro-spinal sclerosis is important; it may be regarded as the type of the so-called *intention* tremors. The affected parts are normal as regards motor symptoms whilst the patient is at rest, but as soon as a muscular movement is attempted the jerky nature of the contractions becomes obvious; there is more or less incoördination and muscular weakness.

Nystagmus, or oscillating tremors affecting the globes of the eyes, is a sign of considerable importance, and is very often associated with the last-mentioned type of tremor. It is of interest to note that it may be a spontaneous tremor like paralysis agitans, or an intention-tremor, only visible upon movement of the eyeballs. It is seen in the majority of cases of disseminated sclerosis, in Friedreich's ataxia, cerebral tumour sometimes, cerebellar disease, ataxic paraplegia, congenital eye-affections, &c., and constitutes a feature in the visual disturbances of coal-miners. (See page 1126.)

SPINAL DISEASES

These will each be found under its own heading, as 'Myelitis,' 'Meningitis,' 'Paraplegia,' 'Ataxia,' 'Syringomyelia,' 'Infantile Paralysis,' 'Muscular Atrophy,' &c. &c.

SPLEEN, DISEASES OF

The affections of this organ are as a rule secondary to morbid processes elsewhere in the body, and they will be found under their respective headings, as 'Leuchæmia,' 'Anæmia,' 'Malaria,' 'Amyloid Disease,' 'Anthrax,' 'Liver Cirrhosis,' &c. &c. *Enlargements* of the spleen, however, occur under such a diverse variety of circumstances that a brief summary of them may be here made for convenience. Of the general affections, in which there is observable a distinct and palpable increase in the size of this organ, the first place, as regards frequency, must be given to *infections*, as *Malta* and *typhoid fever*, *malaria*, *small-pox*, *typhus*, *scarlatina*, *acute tuberculosis*, *relapsing fever*, *pneumonia*, *spotted fever*, *diphtheria*, *erysipelas*, *septicæmia*, *ulcerative endocarditis*, *pylephlebitis*, *pyæmia*, &c. In this group obviously the splenic enlargement is of recent or acute formation, and in one disease at least (*typhoid fever*) its presence is of considerable diagnostic value. In some of the septic conditions, notably *ulcerative endocarditis* and *pyæmia*, and occasionally in *typhoid fever*, the enlargement is due to embolism or thrombosis, and the resulting septic infarcts may break down into pus. The chronic enlargements of the spleen are met with in *leukæmia*, in which the organ reaches sometimes huge dimensions, in the *malarial cachexia*, *cirrhosis of the liver*, *valvular diseases*, *amyloid degeneration*, *Hodgkin's disease*, *syphilis*, *gall-stones*, *pressure* from malignant or other tumours obstructing the portal or splenic vein, &c.

There is a group of splenic enlargements sometimes classified as *primary*; these are usually associated with symptoms of severe anæmia, but as anæmia forms no part of the clinical picture in one variety of these the group is divided into two : (1) *Splenomegaly simple primary*, or *S. without anæmia*, and (2) *Splenomegaly with anæmia*.

1. *Simple or Primary Splenomegaly*.—This is a rare disease, in which there is no sign save great enlargement of the spleen without any obvious cause, and no symptoms except those due to the weight and dragging of the enormously enlarged organ, which may occupy a considerable proportion of the entire abdominal cavity. Several of these rare cases have been recorded in a single family, and their causation is entirely unknown.

2. *Splenomegaly with anæmia* appears in several forms, and these, in the presence of the mystery which surrounds their pathology, are, for the present, retained in one group supposed to include different, though allied, affections.

(a) *Banti's Disease*.—This is a splenomegaly with a very marked degree of anæmia. The blood shows the anæmia to be of the secondary type, with considerable diminution of the red corpuscles, and also of the white, and a low colour-index, with numerous nucleated red cells. The chief characteristic pathological feature in this form of splenic enlargement is the presence of cirrhosis of the liver, which forms, in the later stages of the disease, a typical picture of hepatic cirrhosis, with jaundice and ascites, accompanied by a tendency to hæmorrhages from the stomach, and sometimes from the bowel; deep pigmentation of the skin is sometimes present, and the lymphatic glands are not enlarged. The disease usually runs a slow course for many years.

(b) *Splenic Anæmia*.—This condition is described under 'Anæmia,' upon page 38. The blood-characters, as well as the enormous size of the spleen, the progressive anæmia of secondary type, and the remittent slow course, are practically identical with the clinical picture of Banti's disease, save that hepatic cirrhosis does not appear, with its ascites and jaundice. A condition described as *Splenic Pseudoleukæmia* is probably a modified form of splenic anæmia, and obviously some of these cases are examples of generalised lymphadenoma (Hodgkin's disease), in which the spleen is very large.

(c) *Infantile Splenomegaly*.—This form occurs in infancy or very early childhood, and in addition to the splenic enlargement there is usually, if not always, marked enlargement of the liver, and sometimes increase in the size of the lymphatic glands. The disease runs a very chronic course, and may be associated with hæmorrhagic tendencies, pigmentation, and jaundice. Occasionally recovery ensues, but death from profound anæmia of a well-marked secondary type is the rule. The condition is practically identical with that known as *Von Jaksch's Disease* and *Pseudoleukæmia Infantum*, in which, in addition to a lymphocyte leucocytosis, the blood is found charged with a large number of nucleated red cells.

Tropical Splenomegaly, known as Kala-Azar, is described upon page 1359, under the heading of 'Piroplasmosis.'

Osler has described a number of cases in which there was enlargement of the spleen with remarkable increase in the red corpuscles of the blood, which sometimes numbered up to thirteen millions per c.mm. With these signs were associated those of chronic cyanosis, without any pulmonary condition to account for the lividity. He claims for this strange condition of affairs the recognition of a well-defined clinical entity of unknown etiology, to which the name of

Chronic Polycythæmia with Cyanosis and Enlarged Spleen is applied.

Tumours of the Spleen are comparatively rare. Jepson and Albert have collected thirty-two cases of sarcoma; hydatids and syphilitic gummata are also met with, and in many instances successful removal of the organ has been accomplished. C. A. Powers recently has collected thirty-two cases of *non-parasitic cysts* of the spleen, which he believes originate in a sub-capsular hæmorrhage, and in ten of these splenectomy was successfully performed.

Rupture of the Spleen from injury is not a rare event; sometimes it occurs when the organ is enlarged in malaria and other conditions. The symptoms are those of shock from severe internal hæmorrhage, and the condition demands prompt opening of the abdomen and removal of the organ, after ligation of the splenic vessels and gastro-splenic omentum.

Movable Spleen.—Like movable kidney, this condition may exhibit no symptoms of anything abnormal in some individuals, whilst in others serious nervous disturbances, pain, dragging, &c., are complained of. The degree of mobility explains to a certain extent these differences; thus, a spleen floating about the abdomen at the end of a long-drawn-out pedicle or cord, consisting of the splenic vessels and stretched gastro-splenic omentum is liable at any time to originate serious symptoms of two types. (1) When the vessels in the pedicle have become twisted by the floating organ having been rotated on its own axis during its excursions about the abdominal cavity, sudden severe pain may set in, with serious depression, caused by the organ filling up with blood which distends its capsule; this inflames and forms adhesions to neighbouring organs. When the torsion is acute the gland may rupture or rapidly necrose and death from hæmorrhage result, but if the torsion has been so complete as to entirely shut off the blood-supply the spleen may wither up. (2) Symptoms of a different kind are produced by the floating tumour when it falls down into the pelvis, and by its pressure causes irritation of the bladder, uterus, or rectum, or when it so drags upon the stomach as to interfere with the patency of the duodenum and cause dilatation of the stomach, and even obstructive jaundice. Where the organ has become anchored by adhesions in some part of the pelvis or about its brim, the thin pedicle may act like a band, and cause acute obstruction in a coil of bowel slipping under it. The condition sometimes, though rarely, is part of a general ptosis of the abdominal organs, as seen in Glénard's disease; often it exists without displacements of other organs, when it is supposed to be due to some congenital weakness of its peritoneal attachments. As in the case of the kidney, it is most frequently found in subjects who have borne numerous children.

Diagnosis.—A floating spleen has been mistaken for a floating kidney, ovarian tumour, or even a uterine fibroid. It is necessary to remember that, owing to the constitution of its pedicle, whose partial torsion interferes with the circulation of the blood in the organ, a floating spleen is very likely to form a tumour of considerable dimensions. The chief diagnostic point is the recognition by palpation of the outer convex surface and the demonstration of its notches.

Treatment of Floating Spleen.—This has of late years been successfully carried out by the operation of splenectomy, but when the organ is found to be otherwise healthy it may be advisable to perform a splenopexy, as successfully carried out by Oppel. The operation of removal of the spleen is followed by no visible ill-consequences when the dangers of sepsis and hæmorrhage have been overcome. In healthy individuals, after splenectomy for injury, temporary enlargement of the lymphatic glands has been sometimes observed, and Ehrlich has pointed out remarkable blood-changes, marked anæmia with polymorphonuclear leucocytosis being in a few weeks replaced by lymphocyte leucocytosis and after a year by an eosinophile leucocytosis. Leukæmia of the splenomedullary or so-called myelogenous type is so seldom benefited by splenectomy that Richardson states this condition should be removed from the category of surgical affections.

SPRUE, or PSILOISIS

Chiefly affecting Europeans in tropical or sub-tropical countries, this is a disease characterised by a peculiar diarrhœa, associated with a low form of catarrhal inflammation of the entire digestive tract, best marked in the mouth, pursuing an irregular course, with intermissions and relapses, and often ending in death from great emaciation and anæmia.

Distribution.—The disease is probably to be found in all tropical climates; it is common in Middle and Southern China, Manila, India, Ceylon, the Malay Archipelago, West Indies, some parts of Japan, and Tropical Africa.

Etiology.—The causal factor of sprue awaits demonstration; the various parasites hitherto supposed to be the actual cause of the disease have been proved to be but accidentally present in the motions. A remarkable peculiarity of the disease is that it never affects Europeans till they have resided for several years in the tropics, and this has led Cantlie, who has closely studied the malady, to attribute it to the use of vegetable oils in the food, which produce prolonged irritation of the alimentary mucous membrane. This theory, however, fails to explain those first attacks of genuine sprue which sometimes seize upon healthy Europeans many months, or

even several years, after their return home. Van der Scheer regards sprue as an intoxication, the result of a sequela of appendicitis, having found the distal portion of the appendix occluded in seven out of nine cases investigated.

Natives also occasionally suffer. All ages may be attacked, but the proportion of children affected is small, and women appear to suffer more frequently than men.

Symptoms.—Stephens epitomises the symptoms as detailed by Cantlie: (1) A bare and usually painful state of the tongue; (2) Frequent attacks of stomatitis with ulceration in the buccal cavity; (3) Frequent, soft, bulky, fermenting, pale stools; (4) Anæmia and loss of strength.

The disease commences insidiously, and is generally preceded by frequently recurring attacks of sore mouth, tender gums, and follicular ulcers on the inside of the cheeks; a form of ulcer described by Crombie as occurring opposite the last two upper or lower molars is believed to be a frequent precursor of the disease. Diarrhœa sets in suddenly, or commences gradually, with a tendency towards looseness of the bowels in the early morning hours. The stools vary considerably; the most common appearance is that of a dull yellowish-white colour, of soft consistence, and frothy from the presence of numerous bubbles of gas, which latter appearance alternates with that of stools of the consistence of soft mud, devoid of all evidence of fermentation. As the disease progresses the motions become liquid, foul-smelling, and brownish; they may be tinged with blood, and sometimes mixed with mucus, as seen in dysenteric disease. Scheer regards the *fatty* stool as the characteristic symptom of sprue, and maintains that the disease can be recognised from its onset by a chemical or microscopical examination of the evacuation, though the presence of fat is not due to pancreatic change. This fatty character of the stools is not, however, mentioned by Manson or Cantlie.

With the irregularity of the bowels the other characteristic signs of the disease develop; the stripping of the tongue becomes more marked, the condition being usually described as *psilotic*: it becomes glazed, fissured, ulcerated, and sometimes intensely painful, so that mastication, or even swallowing, is almost impossible. The condition of the tongue, however, in many cases appears to bear no proportion to the diarrhœa, dyspepsia, and emaciation. Fever is usually absent, except in the acute exacerbations, when the mouth is deep-fissured and ulcerated.

When improvement commences this is first signified by a return of the stools to the normal in colour and consistence, and a gradual disappearance of the wasting; but relapses are frequent, and the course of the disease, if not ending in a fatal issue from some complication like pneumonia,

may drag on for years, the patient gradually sinking from marasmus.

Pathological Anatomy.—The changes are in the main those found in death from starvation and diseases causing arrest of assimilation. All the organs are shrunk and wasted, the liver notably so, and this constitutes a valuable physical sign sometimes during life. Manson describes the atrophic wasting of the bowel as causing a thinning so marked as to produce a diaphanous appearance. There are ulcers or erosions in the great intestine and rectum which have led to the erroneous theory that the disease is but a variety of dysentery; ulceration is absent or but marked to a trifling extent in the small intestines, but amyloid degeneration is occasionally observed, and secondary parenchymatous changes are observed in the pancreas and kidney.

Diagnosis.—The disease is only liable to be mistaken for *dysentery*; but the character of the copious pale or drab stools, with the history of the case and the characteristic condition of the mouth and tongue with its denuded epithelial covering and its fissures, ulcerations, or erosions, which often precede the bowel-symptoms, are usually distinctive.

Treatment.—This must be mainly dietetic, and the best routine is to confine the patient to bed and give a milk *régime*, administered in small quantities and very frequently, as in gastric and intestinal ulceration. Cantlie alternates this with *meat* treatment, or gives all through the treatment only pounded beef lightly cooked, beef-juice, soups, or beef-jellies. Several authorities agree upon the value of fresh strawberries and other fruits in the dietary. Some advocate santalin and antiseptics; Hartigan has published results showing the virtues of 3 min. doses of cyllin in capsules; castor oil in small quantity is found beneficial in controlling the diarrhoea. The above dietetic treatment, if perseveringly carried out for several months, reduces the mortality of the disease markedly, and in many instances leads to a permanent cure if commenced before the atrophy of the intestinal mucous glandular apparatus has become established.

STOMACH, DISEASES OF

The various affections of this organ will be found described under their respective titles, as 'Dyspepsia,' 'Gastritis,' 'Gastric Ulcer,' 'Gastric Cancer,' 'Gastrectasis,' 'Gastralgia,' &c.

STOMATITIS

Under this title are included several distinct types of inflammation affecting the mucous membrane of the mouth—i.e. the *Catarrhal*, *Aphthous*, *Ulcerative*, *Parasitic*, and *Gangrenous*.

CATARRHAL STOMATITIS

This is also termed *Acute*, *Erythematous*, and *Simple Stomatitis*.

Etiology.—The affection is most common in childhood, but occasionally adults suffer. The causes may be local or general; the local origin is seen in those cases where there is evidence of irritation arising in the immediate vicinity of teeth about to force their way through the infantile gums, or in adults suffering from dental caries, or after the introduction into the mouth of soiled teats, or irritants, as spices, scalding food, or tobacco. The general causes producing a catarrhal condition of the mucous membrane of the mouth are seen in cases of dyspepsia and intestinal disease, and in scarlatina, measles, and other eruptive fevers. Mercury, iodides, bromides, and other drugs act locally upon the mouth; after being first absorbed into the blood they are excreted by the salivary glands and irritate the mucous membrane, which is constantly soaked in the saliva containing these drugs. Tobacco acts in this way also, in addition to its primary and direct local irritation, whether smoked or chewed.

Symptoms.—These depend upon the extent and localisation of the inflammation, which may be either limited to the tongue, gums, inside of the lips, or may, in rare cases, involve the entire lining of the mouth. At first the appearance is that of an erythematous redness, usually of patchy distribution; there is dryness and considerable tenderness or local discomfort. Later the typical catarrhal stage is reached, in which the mucous surface becomes thin, opalescent, opaque or white from alteration in the epithelium, and there is usually increase in the salivary secretion.

As a rule there are no constitutional symptoms, the condition rapidly yielding to treatment. In some cases, however, slight erosions may occur, leading to lymphatic-gland enlargement, and in a small percentage of cases the catarrhal may be the precursor of one of the other forms of stomatitis.

Treatment.—The inflammatory redness and difficulty in mastication, sucking, or swallowing speedily yields to repeated cleansing of the mouth by a weak boracic-acid solution or, better still and more convenient, by the brushing of a little glycerin of borax over the tongue or lips.

APHTHOUS STOMATITIS,

Known also as *Vesicular*, *Follicular*, and *Herpetic Stomatitis*, is a very common form.

Etiology.—The disease arises from similar causes to those which produce the catarrhal variety, and is most frequently observed in children suffering from gastric or intestinal disturbances. Though a parasitic origin seems highly probable, no specific organism has been demonstrated.

Symptoms.—It appears in numerous small greyish or yellowish-white patches, each surrounded by an erythematous ring or areola. The first stage of the eruption, usually seen upon the inner surface of the lower lip, is papular. The papules rapidly become vesicles, and within twenty-four or forty-eight hours the epithelial covering of each is shed, leaving a small superficial or shallow erosion, somewhat depressed in the centre. They may be few or many in number, and are seen over the dorsum or edges of the tongue, or inside of the cheeks, and may spread to the mucous membrane of the hard and soft palate. There are usually but slight constitutional symptoms, as a mild degree of fever, but the local irritation and pain from the ulcers or erosions lead to great difficulty in taking food; the saliva is always greatly increased, and some enlargement and tenderness of the neighbouring lymphatic glands are commonly present. The condition usually yields to treatment within a week, but when occurring in debilitated subjects the ulcers may remain indolent for several weeks, though owing to their superficial and shallow character no scarring results.

Treatment.—This form of stomatitis also yields rapidly to the glycerin of borax smeared or painted over the tongue or inside the lips. Chlorate of potassium is also efficacious, both locally and internally. Feeding should be carefully attended to, and only liquids or impalpable farinaceous foods should be permitted. When the ulcers are slow to heal and the difficulty of feeding is considerable, each erosion on the tongue may be touched with a minute brush moistened with pure carbolic acid. The solid caustic point is highly efficacious, but very painful.

ULCERATIVE STOMATITIS

This variety is sometimes known as *Phagedænic Gingivitis*, *Putrid Sore Mouth*, and *Fetid Stomatitis*.

Etiology.—Children between four and thirteen or fourteen years are most frequently affected; rarely is the affection seen in infants, and never till after the eruption of some of the milk-teeth. No specific parasitic organism has been demonstrated. Two causal factors are nearly always present, generally in combination—viz. defective hygiene, as seen

in squalid environment or bad feeding, and necrosis of the teeth. When the disease appears in adults it is nearly always the result of mercurialism; the writer has never witnessed this drug produce the disease in infants or young children, but probably moderate mercurial treatment would induce the affection at any age if the kidneys were diseased and the teeth in a bad condition. Mercurial stomatitis commences always as a catarrhal affection, but if the drug be not suspended the ulcerative type is very likely to supervene, and it is not rarely met with in those who have inhaled the vapour of the metal when spilled in laboratories or pharmacies.

Symptoms.—The first appearance of the disease begins in a deep redness and spongy swelling upon the free margin of the gums, especially marked upon the projections of the mucous membrane between the teeth. The region of the molars is most frequently involved, but the commencement of the swelling is usually determined by the site of some sharp, ragged, projecting stump, or about the margins of a hollow cavernous tooth, whose crown has almost disappeared. The spongy gum bleeds readily, and strips from the underlying tooth, and soon necrosis or molecular death of the mucous membrane occurs, forming superficial grey or yellowish-white ulcers, which spread by contact to the contiguous aspects of the inner side of the cheek. The tongue swells, becomes coated and indented, alkaline foul saliva dribbles freely from the mouth, the breath becomes intensely fetid, the teeth loosen and sometimes drop out, and the neighbouring lymphatic glands swell and become tender.

In severe cases a thin film of bone necroses and is separated from the jaw in the vicinity where the teeth have been shed. The constitutional symptoms are always more severe than in the catarrhal or aphthous varieties, more or less fever being usually present, with anæmia. Owing to the pain in mastication and deglutition caused by the swollen or ulcerated condition of the tongue, food is taken in insufficient quantity, and unless prompt treatment is early started there may be wasting. The products of decomposition, caused by the development of pus-forming organisms in the unhealthy mouth are swallowed, and mild toxæmia results, sometimes exhibiting its presence by the appearance of a septic rash. The disease is, however, usually amenable to treatment and runs its course, in the majority of instances ending in recovery within a couple of weeks.

Diagnosis.—The only affection likely to be mistaken for ulcerative stomatitis is *cancreum oris*; but in that disease, which is also known as gangrenous stomatitis, from the first the grave aspect of the ulcerated area, which is situated upon the inner side of the cheek, is obvious by the brawny thickening and induration of the surrounding parts; these rapidly

pass into a gangrene, which spreads with terrible rapidity, destroying a considerable portion of the face.

From *Bednar's aphthæ* the disease is differentiated by the ulcerative process starting in the gums. The affection so named occurs in weak bottle-fed infants, and appears as an ulcerated patch on the mucous membrane covering the roof of the mouth, supposed to be produced by an abrasion through contact of the nurse's finger or the rubber teat of a feeding-bottle. The abraded surface becomes infected by the ordinary organisms present in the mouth, and molecular disintegration of the elements of the mucous membrane leads to localised ulceration.

From *pemphigus vegetans* attacking the mouth the disease is also easily differentiated by its origin in the gum in the neighbourhood of diseased teeth; moreover, in this rare and grave affection the characteristic bullæ of pemphigus soon appear upon the skin in various regions, and deep ulcerations rapidly follow, usually ending in a fatal result.

Treatment.—Chlorate of potassium has a specific action in this form of stomatitis. To obtain its full beneficial effects it should be given internally, and be used freely and frequently as a mouth-wash, after previous cleansing with weak chlorine or permanganate of potassium solution. In less acute cases tabloids consisting of the chlorate, borax, and cocaine may be constantly sucked in the mouth, or left between the molar teeth and the cheek. The drug should be at the same time freely administered in solution, and its best effects are obtained when it is given in combination with the tincture of perchloride of iron. The ulcers may be lightly touched with strong solution of iron or of mercury perchloride, or solid lunar caustic, or liquid carbolic acid. Feeding by fresh meat-juices and other concentrated liquid food is essential from the start; rarely will alcoholic stimulation be indicated save in cases associated with much debility.

PARASITIC STOMATITIS, or THRUSH

This is a common variety of stomatitis, known also by the names of Soor, Muguet, and Mycotic *Stomatitis*.

Etiology.—The condition usually attacks ill-fed infants, in whom the infection is conveyed through the medium of dirty or foul feeding-bottles, and may be transmitted by an infected human nipple to several infants applied to the breast of a mother whose infant is suffering from thrush—a habit not uncommon amongst the poor. The disease is very often seen in adults suffering from the later stages of phthisis, long-continued fevers, cancer, &c.

The causal factor is the presence of a widely distributed fungus known as *oidium albicans*, *saccharomyces albicans*, *oidium lactis*, &c. Some authorities recognise these as different

varieties of fungi, whilst others regard the variations of form and spore-formation as mere developmental peculiarities dependent upon the culture-medium. The fungus in its growth exhibits long unequal filaments, with a refractile ovoid cell or nucleus at each end; these flourish in the layers of the epithelium, but do not penetrate into the deeper mucous-membrane constituents, and may be readily recognised under the microscope in a superficial scraping from one of the white patches. This shows a dense network of mycelial threads, oval cells, epithelial *debris*, leucocytes, and various mouth-bacteria.

The existence of catarrhal stomatitis from any cause affords a suitable nidus for the growth of the fungus, which probably is unable to develop upon a healthy mucosa.

Symptoms.—The disease appears generally upon the dorsum of the tongue or inside of the cheeks and spreads over the entire buccal lining in the form of small white flecks resembling specks of curdled milk. These increase in size and, coalescing, form variably sized patches, or in rare cases one continuous sheet of white membranous exudation, which may cover the pharynx and extend down the gullet. The patches are quite superficial and can be readily detached by gentle scraping; usually a bleeding or oozing base becomes visible after the separation of the felted epithelial layer. Not infrequently some erythematous redness may be detected in the mucous membrane surrounding the patches, showing either a previous or a secondary catarrhal stomatitis, but otherwise the mucosa is normal.

The mouth is generally in a drier condition than the normal, and Still draws attention to the fact that the saliva is almost invariably acid in reaction—a point in contradistinction to the alkaline salivary secretion which is characteristic of the ulcerative form of stomatitis.

The fungus is believed by some authorities to pass down the intestinal tract and develop about the anal aperture; but proof of its presence here or in the excoriations upon the inside of the buttocks or genitals which so commonly accompany the diarrhœas of infancy is wanting, though its presence in the stomach has been demonstrated.

Often constitutional symptoms are entirely absent; when such are present, they are usually due to the primary malady of the stomach or bowels which has caused the debilitated condition favouring the growth of the fungus in the mouth.

Usually the disease is brought under the notice of the nurse by some trifling difficulty observed in the infant whilst in the act of feeding. The condition seldom, however, leads to any serious difficulty in deglutition, and usually passes off in a few days under proper management.

Treatment.—Absolute cleanliness is an effective preventive, and when the disease has already appeared in a mild form nothing is needed in breast-fed infants but to smear over the mother's nipple with the glycerin of borax before the child is put to the breast. In bottle-fed children the sterilisation of feeding-bottles and rubber teats should be carefully seen to and the mouth gently swabbed out by a piece of lint soaked in glycerin of borax, which is a more reliable preparation than the old *mel boracis*. When the pharynx is involved, a spray of boric acid is indicated.

GANGRENOUS STOMATITIS

This is described under its more common and ancient name of '*Cancrum Oris*' upon page 159. Owing to its serious nature and high mortality, the importance of early differentiation from the more innocent varieties of stomatitis cannot easily be exaggerated, especially since the only hope of successful treatment lies in the early application of radical surgical measures. In a few cases the disease has been known to supervene upon mercurial stomatitis, and even to have been preceded by the ordinary ulcerative variety.

SUDAMINA. (See under *MILIARIA*, p. 1044.)

SUNSTROKE, or INSOLATION

This is the name given to a pathological condition arising from exposure to excessive heat.

Etiology.—Little need be said under this head, since the causative factor is obvious; but several predisposing and modifying influences should be kept in mind. In the first place, it is hardly necessary to emphasise the fact that, though the most frequent cause of sunstroke is exposure to the direct solar rays in the daytime, the condition also occurs in persons exposed to solar heat in the shade and at night. Moreover, exposure to artificial heat acts in a similar manner; hence the use of such terms as *Thermic Fever*, *Heat-fever*, *Heat-apoplexy*, *Heat-stroke*, *Heat-shock*, *Heat-exhaustion*, &c. but these terms are held by different writers to signify in some cases distinct conditions: thus *heat-exhaustion* differs in some respects from typical sunstroke. It is, however, advisable to regard all the forms as varieties or degrees of a condition produced by exposure to heat.

Children and women, probably from their less frequent opportunities of exposure, suffer less than adults and males; Europeans are more susceptible than the dark-skinned races,

newcomers especially being liable to attack in tropical climates. The condition is not, however, confined to the tropics, and is seen occasionally in the hot summers of temperate latitudes. The frequency of sunstroke, moreover, does not necessarily depend upon the mere height of the temperature usually recorded in a given locality, being intensified by moisture, a low-lying position, stillness of the atmosphere, the proximity of stagnant swamps, leading to the accumulation of impurities in the air, and many other climatic and meteorological conditions.

Of the personal factors which render individuals susceptible, muscular exertion and alcoholic excesses are powerfully acting causes, so also are over-eating and over-clothing. A large proportion of the deaths from sunstroke occur in armies on the march, but the substitution of lighter accoutrements and more sanitary trappings have lessened the frequency of attacks considerably in later years. The condition is, however, still frequent amongst the stokers employed in steamships, and is not uncommon in ill-ventilated barracks; and sometimes it occurs at sea during the night in crowded cabins or cramped ill-ventilated sleeping-berths.

Symptoms. — *Heat-exhaustion.* — In a *mild degree of sunstroke* the only symptom may be a transient syncope: the heart fails, some degree of unconsciousness supervenes, and the usual accompaniments of syncope are evident, as pallor, sweating, dilatation of the pupil, normal or sub-normal temperature, and hurried respiration. This constitutes the simplest type of what is commonly known as heat-exhaustion, or the mild cardiac type of sunstroke. The attack usually follows exertion in the sun, but it may occur at night or in the shade, may come on suddenly or be preceded by giddiness or by a sensation of faintness. The patient usually recovers rapidly upon the application of judicious treatment, a certain degree of prostration or physical depression only remaining, with or without headache.

In a more severe attack the syncope may be fatal, but usually other signs are then present and the previously mentioned symptoms deepen: the pallor and collapse become extreme, the temperature may fall to 95° , and the respirations may stop along with the heart. In either case, when the pulse has not disappeared it is very feeble, small, and rapid. If consciousness does not speedily return, and in some cases where it has been retained throughout or after it has been regained, the condition passes from that of heat-exhaustion into that of *thermic fever*. The pallor gives way to flushing and the temperature rises rapidly.

Thermic Fever. — In a typical *severe attack of sunstroke*, usually recognised as thermic or heat fever, there may be prodromata of giddiness, nausea, headache, visual disturb-

ances, frequent micturition, anxiety, restlessness, dyspnœa, insomnia, &c. Often, however, the attack comes on without a moment's warning, and in rare cases it may prove fatal within less than half an hour.

The symptoms vary considerably according to the part of the organism which receives the brunt of the attack, but always there is complete unconsciousness, eventually amounting to profound coma, and an exalted temperature which rapidly reaches hyperpyrexia, the thermometer registering often 110°. The pulse is usually quick and weak, though it may later on become slow, full, or markedly irregular. Dyspnœa is present, the quick shallow respirations passing into the Cheyne-Stokes type, or marked stertor sets in. The appearance of the face contrasts with that seen in heat-exhaustion: the cheeks and neck are flushed and become livid, the conjunctivæ are deeply injected; the pupils at first dilate, but as the attack develops they contract to a point, and widen again before death, and the light-reflex is usually absent. The tendency is towards death from asphyxia and heart-failure. Mucous rattles are heard as in ordinary apoplexy, and as the blood becomes highly venous epileptiform convulsions occur. The urine is suppressed, or the scanty secretion may be discharged along with the bowel-contents in the relaxation which precedes death. The entire skin may be deeply livid, and petechiæ are not uncommon.

From the profound coma, which is always in marked contrast to the fleeting syncopal unconsciousness of heat-exhaustion, generally the patient cannot be roused at any stage of the attack in the grave cases. But when prompt and judicious treatment is at hand the temperature falls, the breathing becomes less laboured, the light-reflex returns, twitchings or subsultus pass off, and gradually the patient returns to consciousness. The duration of the attack may vary from one to four hours to as many days. From the severe attacks, though recovery is possible, it is seldom complete: the individual is never the same as before; his mental powers are weakened, memory is feeble, epilepsy may supervene; emotional instability, constant headaches, impaired vision, deafness, or paralysis the result of cerebral hæmorrhages which are liable to occur at the acme of the original attack, may remain permanently. It is not rare for a condition of total dementia to supervene in severe and protracted cases, and sometimes insanity develops long after the attack.

Even when recovery is apparently complete, nearly all the subjects of sunstroke show a remarkable susceptibility to moderately high temperatures; they are liable to attacks of extreme restlessness and headache during the warm summers of temperate climates, and they become markedly

excited by moderate doses of alcohol which formerly produced no appreciable stimulation. In others a condition resembling that observed in general paralysis of the insane may be observed, but, as pointed out by Macleod, though the difficulty of articulation and a considerable degree of muscular paresis is obvious, there is no exaltation or morbid euthesia present.

Morbid Anatomy and Pathology.—The blood is found dark or almost black, venous congestion of the lungs is extreme, and general venous stasis is observed, with an empty state of the arterial system. The blood has been found to give an acid reaction; it does not coagulate; its red corpuscles are crenated and do not form rouleaux. The heart is usually found firmly contracted, and more or less cerebral congestion is present, though extravasations or other organic lesions are rare. Rigor mortis sets in very early and putrefactive changes occur rapidly.

In subjects who succumb to heat-exhaustion the changes are much less obvious, sometimes the only abnormal condition being a flaccid heart, whose right side is distended with dark fluid and venous blood or a soft black clot, whilst the left chambers are empty; and the brain may show no evidence of congestion, though the abdominal viscera may be livid and the veins distended. The interpretation of the changes, especially those seen in the cardiac organ, is difficult. By many authorities the sudden death in the acute cases is believed to be due to the coagulation of the cardiac myosin, which has been demonstrated by Sir Lauder Brunton to occur in animals exposed to intense heat, though Wood regards this coagulation as a *post-mortem* phenomenon. Death in most cases which are not of extremely short duration is obviously due to hyperpyrexia, brought about by disarrangement of the working of the heat-centres caused by the exposure to a temperature considerably above that of the normal blood. There is probably increased heat-production with diminished dissipation. The theories of a microbic or toxæmic origin are not supported by facts.

Diagnosis.—The presence of distinct hyperpyrexia without any of the symptoms of the diseased conditions in which very high fever-heat is met with is practically pathognomonic when taken in association with the history of exposure to a heat about or above that of the blood. The only difficulty in the diagnosis arises in those rare cases where a patient suffering from an affection like acute rheumatism or malaria is at the same time exposed in a tropical climate to an atmosphere where the thermometer registers a considerable figure above blood-heat, or at all events above 95°. The history in such cases should leave little doubt, though it may be probable that a patient already feverish might become suddenly hyper-

pyretic during an exposure to a temperature which would not cause sunstroke in a healthy individual.

From the coma of *alcoholism*, *uræmia*, *opium*, or *apoplexy* the exalted temperature alone is sufficient to establish the diagnosis. Some of the acute fulminant cases occurring in the recent Belfast epidemic of cerebro-spinal fever bear a close resemblance to sunstroke, but even in the most fulminant or apoplectic type of the disease the hyperpyretic temperature characteristic of sunstroke is never recorded.

From *heat-exhaustion* (which is, however, but the mild syncopal form of sunstroke) the typical form of sunstroke is easily differentiated by the thermometer. In heat-exhaustion the temperature is not elevated and the loss of consciousness is transient and often incomplete, the face is pallid, and the skin moist, as in most cases of ordinary collapse from whatever cause.

Prognosis.—The grave form, with very high temperature and absence of the light-reflex, with symptoms of asphyxia, has a very high rate of mortality. The degree of consciousness is sometimes a rough criterion of the gravity of the case, the prognosis being obviously bad where the coma is progressively deep and the asphyxia obvious.

Heat-exhaustion has a better forecast, the great majority of the cases ending in complete and speedy recovery; but an apparently mild case of heat-exhaustion is sometimes seen to pass into a hopeless syncope; whilst, on the other hand, sometimes a profound coma, associated with hyperpyrexia, yields to energetic treatment even after one or two days of grave anxiety upon the part of the physician. An alcoholic history deepens the gravity of both conditions.

Treatment.—Preventive measures are obvious. The errors in clothing, especially those which cause constriction about the neck and chest, should be rectified. It is necessary that the spine and the cranium should be well protected from the direct solar rays. Violent exercises in hot weather must be avoided, over-eating and alcoholic excesses should be strictly forbidden, and the freest possible ventilation ensured in the day and night rooms of the house.

When an attack of heat-exhaustion has already occurred the ordinary measures indicated in syncope are to be employed. The patient should be carried immediately to the nearest shade and maintained in the horizontal position with the head lowered, and all clothing about the upper portion of the body rapidly removed. As the patient is at first usually unable to swallow, strong ammonia or acetic acid should be applied to the nostrils or a large dose of alcohol given in an enema and artificial respiration performed.

In a severe attack of thermic fever, the above preliminaries having been rapidly accomplished, the patient should be

freely douched with cold water or plunged into an ice-bath, or wrapped up in a sheet wrung out of cold water and a small stream of this kept trickling over the cold pack. The rectum may be flushed out with iced water, and sometimes it may be more convenient to rub the entire cutaneous surface with large pieces of ice or with handfuls of powdered ice, if available.

The thermometer should be used almost continuously, and as soon as the temperature has been brought down to a little above 100° the application of cold should for the time be suspended.

In the presence of severe asphyxia a vein should be opened without hesitation. Threatened heart-stop may be met by cardiac stimulants or rapidly acting tonics like strychnine; but their routine-use cannot be free from danger, considering the state of the cardiac muscle usually found after death. It is safer to rely solely upon artificial respiration. When convulsions occur chloroform should be administered, and, if at hand, nitrite of amyl. The ice-cap, combined with sinapisms to the nucha, is the best routine for the severe headache or threatening meningitis. After recovery, a quiet existence in a northern or temperate clime and the absolute avoidance of alcohol should be recommended.

SYCOSIS

This affection is described under 'Ringworm,' page 1538.

SYNCOPE

This condition, which is one dependent upon cerebral anæmia, is a symptom of many heart-affections, and is also frequently the result of hæmorrhages, pernicious anæmia, &c. The condition is allied to Shock and Collapse (see page 1597). Local syncope is described under 'Raynaud's Disease,' page 1493.

SYPHILIS

This is a specific infectious exanthematous fever due to the presence of protozoan organisms conveyed by contact or inoculation and by hereditary transmission, characterised by an incubation-stage during which a local lesion develops at the site of inoculation, a secondary or eruptive stage of variable duration, followed often by late sequelæ or tertiary manifestations, invading the viscera, muscles, bones, skin, and nervous system.

The history of syphilis is enveloped in a considerable degree of obscurity, the disease being first recognised as a distinct affection towards the end of the fifteenth century (1495), when it appeared almost in epidemic form in Naples during an invasion by the army of France, being supposed to have been imported a short time previously from the West Indies by the sailors of Columbus. It is, however, almost certain that the disease had long existed in China, and probably in India and even in Europe, previous to this date. Dabry states that the Chinese recognised syphilis more than 2000 B.C. and treated it by mercurial inunctions, but his conclusions from the Hoang-ti writings are seriously questioned.

Etiology.—The disease is invariably produced by the admission of a virus which has the power of reproducing itself in the blood and tissues. This virus, excluding the hereditary form of the disease, is usually transmitted during sexual intercourse through some minute abrasion upon the mucous membrane of the genital organs; though it is probable that infection or inoculation so-called can occur without any breach of surface or excoriation, the living germs effecting an entrance into the lymphatic vessels through the unabraded delicate epithelial lining of the mucosa. The transmission not infrequently occurs at other sites, as on the lips and tongue, the virus being conveyed in the act of kissing or by the use of drinking-vessels or eating-utensils infected by a syphilitic patient suffering from sore mouth. The finger of the accoucheur is probably never infected, unless it has been wounded during an operation or has been the site of a previous wound or marked abrasion.

Individuals of all ages and all races are liable to contract the disease, though the severity of the attack varies greatly amongst different nationalities and races. One attack confers immunity for a long time, usually for life; when a second infection develops into syphilis in a previously infected subject the disease is always of a very modified character, and Hutchinson has pointed out that second inoculations usually produce a soft or abortive chancre.

A very important subject is that of the relative infectiousness of the different stages of syphilis. Whilst it is accepted that the infection from the primary sore and the early secondary lesions is most active, the generally acknowledged view is that the tertiary lesions are sterile; but recently C. M. Williams has produced cogent reasons why the secretions from these latter should be considered as capable of transmitting the disease. Especially significant is the fact that, though experimental inoculation of the products of gummatous growths formerly failed to produce syphilis, the disease has recently been successfully transmitted to apes by inoculating the material obtained from the *spreading margin* of the gumma.

It has been abundantly proven that the virus from a case of congenital syphilis can transmit the disease to a healthy wet-nurse, though the mother of the infant, showing no signs of the disease, is immune to nipple-infection, as seen in Colles' law.

The nature of the syphilitic parasite has long eluded detection ; it has for a considerable period been surmised to be a protozoon. The old view of the *duality* or *plurality* of syphilis need not be discussed : that many different species of organisms may be mixed with the specific virus of the disease at the time of inoculation is obvious when the localisation of the sores is considered. It seemed highly probable that the non-infecting chancres (*i.e.* those whose presence is not followed by constitutional symptoms of syphilis) were due to either an attenuated and sterile syphilitic virus or to pyogenic organisms. The point, however, has been set at rest by the discovery of Ducrey, who demonstrated the specific bacillus of the soft sore in 1889.

The results obtained by Roux and Metchnikoff after inoculating the syphilitic virus into the chimpanzee, when considered in conjunction with recent bacteriological investigation, directly establish the essential unity of the disease.

The search for the specific organism of syphilis has been keenly pursued by a host of observers during the last few years, and the literature of the bacteriology of syphilis has assumed large proportions. A masterly *résumé* of the entire subject is given in the 1907 'Medical Annual,' by J. W. T. Walker, with microphotographs from Fanoni and MacLennan. The discovery of *spirochætae* by Schaudinn and Hoffmann in the various lesions of syphilis marks a new epoch in the study of the etiology of the disease. Two forms are described: One *spirochæta refringens*, is acknowledged to be a saprophyte, bearing no causal relationship to syphilis; the other, designated *spirochæta pallida*, *treponema pallidum*, and *spiromena pallidum*, is maintained to be the true germ of the disease. It is a flagellate protozoon in the form of a thin spiral or corkscrew-shaped actively motile filament, pointed at both ends, and measuring in average length about the diameter of a red blood-corpuscle, with many twists or regular spirals. The organism stains rose-pink with Giemsa's stain. It has been found by many other investigators in chancres, in lymphatic glands, in the blood, spleen, liver, mucous patches, skin-papules, &c., in acquired syphilis and in the various lesions of the hereditary form, and sometimes also in tertiary lesions. It is a very significant and important fact that Metchnikoff and Roux detected the *spirochæta pallida* in the chancres in four out of six inoculated apes.

The question of whether this protozoan body is the true cause of syphilis cannot be said to be finally settled ; those

who still hesitate to accept it as such point out that its numbers in a given specimen bear no proportion to the severity of the lesion, being most plentiful in organs not apparently attacked by the syphilitic virus and most scarce in those where the infection seems most active. Maclellan's conclusion will probably turn out to be correct; he believes that the spirochæta is but a stage in the development of an organism which in all probability is the cause of syphilis, since the slides sometimes swarm with other forms, which he regards as developmental stages of the protozoan parasites or of *Cytoryctes luis*. Marshall accepts the parasite as the causal factor, and states that the only fact wanting in the demonstration is the failure in cultivating it outside the body. Metchnikoff is so satisfied about its specific nature that he defines syphilis as 'a chronic relapsing spirillosis.' At this stage of the investigation of the relationship of spirochæta pallida to syphilis Bing's results are most significant; he found that the parasite rapidly disappears from the lesions after the administration of mercury. Most observers have corroborated the earlier statements that the parasite disappears in syphilitic lesions a few hours after their removal from the body.

The relationship of syphilis to yaws will be referred to under the latter name.

Stages and Symptoms.—The diseased process is usually divided into the three well-marked stages of *Primary*, *Secondary*, and *Tertiary* syphilis. To these may be added the *Parasyphilitic* or *Quaternary* stage, in which, long after the disappearance of the living virus, certain degenerative changes of the nature of sequelæ sometimes manifest themselves.

PRIMARY STAGE.—The local sore or chancre appears at the site of inoculation, which is commonly the glans or prepuce in the male, on an average of about twenty-five days after coitus or after experimental inoculation. These twenty-five days constitute what is called by Marshall the first incubation-period. After about double the length of this period from the first appearance of the sore—viz. in forty to fifty days—the second incubation-period terminates with the rash or other symptoms of the invasion as seen in the general or constitutional infection. The total incubation-period of syphilis is therefore upon an average about seventy days, markedly contrasting with the corresponding incubative period of thirteen days in small-pox. The contrast is still greater when we look at the length of the period of invasion of the so-called secondary stage, which varies from about one and a half to two years.

The primary sore which constitutes the local lesion of syphilis must not be regarded as a localised manifestation of the disease; by the time the sore has appeared the blood and tissues have already been invaded by the spirochæta, and complete and wide excision of the local lesion and the

indurated proximal glands has no effect whatever upon the development of the constitutional symptoms. It seems certain that these might appear at their normal time without any local sore having formed, had the virus been introduced into a blood-vessel, as in the cases reported by Jullien and commented on by Marshall, where syphilis followed in two surgeons after inoculation while operating upon an infected patient. Thus it is strictly correct to regard the incubation-period of syphilis as dating from contact till the advent of the secondary period, the incubation-period being broken in upon during its fourth week by the appearance of the sore.

The primary stage is, however, usually counted as commencing with the sore and ending with the eruption; it is identical with the second incubation-period of about forty-five days, and before its termination the sore is usually entirely healed. The typical syphilitic sore presents little tendency towards suppuration or ulceration; its first appearance is a small papule, which soon becomes eroded, and usually covered by a thin scab or crust, underneath which a greyish exuding surface may be found. Its base becomes markedly indurated and of cartilaginous hardness usually within four or six days, and the size of the sore bears no relation to the severity of the after-symptoms. This constitutes the so-called true Hunterian or indurated chancre, with which there is also an enlargement of the proximal lymphatic glands. These, known as the syphilitic bubo, present the same indurated cartilaginous features of the sore, and show no tendency to suppurate, unless pus-forming cocci have become mixed up with the syphilitic virus, and they remain of cartilaginous hardness for a considerable period after the healing of the chancre has been complete.

SECONDARY STAGE.—This, as in the other exanthematous fevers, is ushered in sometimes by prodromata of malaise, febricula, and headache, but as a rule these are seldom obvious till the simultaneous advent of the eruption. The nature of the rash varies widely in different individuals; it may be a bright eruption—roseolar, papular, erythematous, eczematous, pustular, scaly, or pemphigoid in some-cases. There is generally a tendency towards polymorphism—viz. the eruption may be roseolar in one region, whilst in another it has the characters of a measly rash, and shows discrete papules somewhere else. The general tint of the exanthem is that of a smoked ham or copper-colour; it is always more or less symmetrically distributed, in marked contrast to the tertiary skin-lesions, and, unlike these, only the superficial layers of the skin are involved and there is no tendency towards ulceration, unless in very moist parts of the skin, and pruritus is usually absent. Whilst the macular or roseolar and the papular syphilides are practically typical of the secondary stage, the squamous or pustular

may also occur ; the latter in the form of rupia is typical of the tertiary syphilide. With the skin-eruption usually there occurs some invasion of the visible mucous membranes ; the most common site is the tonsils. These may be involved in a deep erythematous red rash which covers the inside of the cheeks, the soft palate, and pharynx ; or this erythema of the pharynx may be present whilst the mucous membrane over the tonsils shows ulcerations of the snail-track type, or it may be the seat of a coating resembling that seen in diphtheria. Apart from the diffuse early redness of the throat, which appears at the commencement of the secondary stage, the chief mucous-membrane lesion is the *mucous patch*. This is to be regarded as the equivalent of the skin-exanthem, modified by the greater degree of vascularity, moisture, peculiarities of epithelial stratification, &c. These mucous patches appear upon the tongue, the tonsils, inside of the lips, corners of the mouth, palate and buccal surface, and sometimes on the pharynx. Upon the tongue the sodden epithelium, especially upon the posterior part of the dorsum, is heaped up and raised considerably above the level of the surrounding non-inflamed mucous covering, constituting the *condyloma*, which is common also about the margins of the anus, on the female genitals, and under the male prepuce. Erosions, fissures, and ulcerations are liable to occur in any part of the mouth from necrotic changes in the mucous patches, but sometimes a hypertrophic epithelial growth or papilloma, akin to the condylomatous thickening on the tongue, is observed ; and all these modifications may be found in the same subject, the mucous exanthem exhibiting the same tendency to polymorphism as the skin-eruption. Occasionally an atrophic condition, without ulceration, occurs in the tongue-papillæ, causing dry denuded patches of various sizes. Local irritation caused by carious teeth, tobacco-smoking, &c., is liable to lead to more or less deep ulceration of the tongue, but the most typical ragged ulcerations are commonly seen about the pillars of the fauces or, as already observed, on the tonsils, and for the most part the mouth-lesions, unless when intensified or determined by the proximity of foul teeth, are symmetrically disposed. The mucous patches contain the spirochætæ in their most active state, and the secretions of the mouth are highly infectious. The cervical and suboccipital lymphatic glands become enlarged and indurated.

The duration of the skin-eruption is most variable ; it may, in mild cases, assume the form of a very transient roseola, which, however, is liable to recur ; but, in the majority of cases, the exanthem continues to develop for three or four weeks, after which it begins to decline and, under vigorous treatment, usually disappears a couple or three months later. It is

not uncommon for the rash to reappear when treatment is suspended at any time during the first year. During the period of eruption slight fever is nearly always present in the evenings; anæmia, headache, some emaciation, anorexia, and a feeling of general malaise, with joint or periosteal pains, are usually also present. The hair often gradually falls out, uniformly over the scalp and beard, but more frequently in coalescing patches of irregular distribution; but permanent baldness is rare, and the nails are seldom seriously affected.

About the declining stage of the eruption symmetrical *iritis* is not infrequently observed—*i.e.* before the end of the sixth month; there is pericorneal injection, the iris becomes thickened and congested, the pupil is contracted, irregular pink nodules of plastic lymph are visible in the anterior chamber, and the pupil does not respond readily to light. Retinitis or choroido-retinitis sometimes accompanies or follows the *iritis*, and a degree of scleritis or episcleritis is also not rarely present. Optic neuritis, papillitis, and atrophy of the disc, when they do occur, are later manifestations.

In many mild cases only a few of the above symptoms are observed, and occasionally the rash escapes the patient's observation, especially when the primary sore has been so insignificant as not to arouse suspicion. At this stage there may be still detected, upon careful examination of a patient first coming under observation, some remnants of induration around the cicatrised chancre, a shotty hardness of the groin-glands on one or both sides, some induration of the cervical glands and snail-track ulceration on the tonsils, and some faint, copper-coloured papules which have succeeded upon the disappearance of the first roseolar eruption.

Within a year of the appearance of the primary sore there is usually very slight evidence of secondary lesions remaining, and as a general rule, even in severe cases, after eighteen months all evidence of the disease has vanished, though recurrent eruptions and buccal mucous patches may, in rare cases, afterwards for years appear at intervals, constituting the group of manifestations called *late secondary syphilides*. These latter in such cases occur, therefore, during the tertiary period, but they can be distinguished usually from the typical tertiary lesions by their susceptibility to mercurial treatment, their tendency towards symmetrical distribution, the frequency of their recurrences, and their potentialities of infectiveness.

TERTIARY STAGE.—This, in many cases, is entirely absent, no further manifestations of the disease being observed after the disappearance of the secondary symptoms, especially when treatment by mercury has been judiciously and most persistently followed.

No definite date can be fixed as marking the advent of the tertiary period, and no hard-and-fast line can be drawn in

many instances between the secondary and tertiary lesions, since, as just mentioned, manifestations answering in most respects to typical secondaries are observed to recur in some patients during many years after the advent of the disease. Nevertheless there are several well-marked and broad distinctions which can usually be relied upon in discriminating these affections:

In a typical case of syphilis, after the total disappearance of the rash, mucous patches, iritis, &c., say by the end of one year or eighteen months, there follows a latent period during which the patient is apparently quite well and free from all traces of his disease. This period may be of only some months' duration, since tertiary lesions have been known to appear as early as the end of the first year, though such an event is very rare. More frequently the advent of the tertiary period is somewhere about the end of the third or beginning of the fourth year of the disease, though it may be delayed for many years, even for a quarter of a century in rare instances.

The old view was that the manifestations of the tertiary period differed essentially from the secondary lesions in their sterility, the virus having perished in the meantime in the patient's blood and tissues, but the recent discovery of spirochætae in the actively extending margins of tertiary lesions, and the successful inoculation of the virus into apes, render this view no longer tenable.

The following broad characteristics of the tertiary lesions are usually recognisable. They show a marked tendency in the new-growth towards degeneration, fibroid proliferation, and ulceration, whilst secondary lesions tend completely to resolve themselves; the latter are almost always superficially situated, if we omit the slight periosteal swellings, whilst tertiary lesions are usually deeply seated, involving bones and viscera, and recurrences and relapses are frequent. The secondary manifestations are more or less obviously symmetrical in their distribution, and generally diffused, whilst many of the tertiary lesions are circumscribed or solitary, and if multiple they are very rarely symmetrically distributed; they are, moreover, monomorphous, and their ulcerations are usually serpiginous in outline. Hutchinson states that the more prolonged and severe the secondary stage is, the greater the risk of tertiary symptoms, whilst, upon the other hand, the figures of Fournier show that in about 90 per cent. of cases, tertiary symptoms followed secondary symptoms of a mild type—a fact satisfactorily explained by Marshall as due to the liability of most patients neglecting treatment when the secondary symptoms are slight.

The lesions of the tertiary period involve the skin and mucous membranes about as frequently as they invade the

different parts of the nervous system; the bones, muscles, testicles, larynx, viscera, and other structures suffer also, but much less frequently.

Skin-lesions.—The new-growth invades usually the entire thickness of the skin or mucous membrane; its cellular elements, the product of a low form of inflammation, tend to break down and cause deep ulcers which, healing in the central portions, extend at their margins, producing the characteristic ringed, circinate, or serpiginous sores so commonly observed in the skin during the tertiary period.

These nodules of the syphilitic granuloma, when they remain circumscribed in the skin, in their advanced developmental stage, constitute a group recognised as the *cutaneous gummata*. They vary in size from that of a haw to a walnut, and may be single or multiple. At first dense in consistence, they soon soften and break down, leaving deep ulcers with punched-out margins, whose bases show a yellow caseous surface or core which may extend down to the periosteum of the underlying bone. *Subcutaneous* or *cellular gummata* are also commonly met with, especially in women, and occur most frequently in the legs, just below the knee. As these indolent hard masses begin to soften they become doughy and convey a false sensation of fluctuation; the skin, which is at first quite movable over their summits, becomes involved and somewhat tender, reddens, and finally ulcerates. The resulting gummatous ulceration reveals the same yellow, wash-leather, caseous core, only usually situated much deeper down in the tissues than in the case of the cutaneous gummata, and, like the latter, they may be multiple or solitary; even if multiple they are not likely to show any tendency towards marked symmetrical distribution.

The *mucous membranes* show similar gummatous ulcerations; the tongue, palate, tonsils, pharynx, and laryngeal mucous membrane are liable to extensive destructive necrotic processes, and the rectum is not rarely also involved. These ulcerations differ markedly from those seen in the secondary stage; they are asymmetrical and deep, and accompanied by little or no inflammatory action. The process being essentially a necrotic one, wide and deep destruction of the parts follows, leading to deformities or stenosis, in which the pharynx, œsophagus, larynx, trachea, or rectum may be partially occluded. The underlying bones and cartilages also often suffer, but there is usually a characteristic freedom from implication of the neighbouring lymphatic glands which is highly diagnostic of tertiary syphilitic thickenings and ulcerations of the skin and mucous membranes.

The *muscles* suffer comparatively seldom, but the tongue-muscles not infrequently are the seat of ulcerative gummata or of a sclerosing process, in which the organ finally

shrinks from the contraction or condensation of the cicatricial tissue.

Bone-lesions.—The bones are often involved; they come next in frequency to the skin, mucous membranes, and nervous system. As already seen, a slight transient periostitis is very common in the secondary stage, causing the so-called osteocopic pains of syphilis, but nodes do not form and resolution is always speedy. The most common bone-manifestation in the tertiary stage is also periostitis, which signifies its presence by the appearance of large tender nodes or gummata, chiefly upon the fronts of the shins, the cranial bones, clavicles, sternum, and ribs, but even the vertebræ may not escape. These nodes may break down and suppurate, leading to caries and the separation of worm-eaten sequestra. Syphilitic caries most frequently attacks the cranial bones; the writer has seen it appear as late as twenty-five years after the primary attack, in a case where a second infection could be certainly excluded, and a very large area of one parietal was shed in this case. The nodular swellings on the cranial bones are sometimes associated with serious cerebral or mental disturbances. In some cases the bone-mischief is situated deeply, the periosteum escaping; this is recognised as syphilitic osteitis, and its only manifestation may be a severe ostealgia, which usually occurs in a long bone in the neighbourhood of a joint. In 1876 the writer witnessed this condition in a patient under the care of Sir John Simon, in whom Mr. Wagstaffe amputated the thigh above the knee for an uncontrollable pain in the head of the tibia which was rendering the patient's life unendurable. The only change found in the bone consisted in a few ill-defined areas of osteosclerosis, deeply situated in the head of the tibia. The condition known as *rarefying osteitis* supervenes when a gumma caseates in the centre, enlarging the Haversian canals and causing a condensation of the osseous tissue at the periphery.

Though the most common form of tertiary bone-lesion is the periosteal node, this probably always should be regarded pathologically as an osteoperiostitis, since the process appears invariably to extend into the true osseous tissue. The joints are seldom affected, though in the secondary stage arthritic pains are common; they are, however, not rarely seen to be transiently involved in the congenital affection in which Mr. Hutchinson describes effusion into the knee-joints coincident with keratitis.

Visceral lesions.—These are as a rule late tertiary manifestations; the liver suffers more frequently than any other abdominal organ. Three well-recognised forms are met with—viz.: (1) Circumscribed gummata. (2) A more or less diffused fibrosis or interstitial hepatitis in which there is marked

increase in the fibrous tissue of the organ, causing a condition resembling cirrhosis. (3) The remaining form is usually associated with the second; in it the capsule of the organ undergoes considerable thickening, the condition being recognised as syphilitic capsular hepatitis. The amyloid degeneration found in the liver, spleen, and kidney does not occur in the tertiary period, as usually defined, but is regarded as a quaternary or parasymphilitic sequela. A malignant form of jaundice has been sometimes observed in the secondary stage which bears a close relationship apparently to acute yellow atrophy.

The kidney suffers more frequently than is generally recognised; according to the statistics of Karnoven, one in every four syphilitic subjects shows *post-mortem* renal disease. In the secondary stage an acute nephritis very occasionally supervenes; in the tertiary there may be gummata or parenchymatous conditions practically identical with the ordinary types of chronic Bright's disease, and in the parasymphilitic stage amyloid disease is liable to supervene.

Testicular lesions.—These are very common; and since they occur amongst the earliest of the tertiary manifestations they were formerly regarded as late secondary lesions, though Marshall states that they seldom appear before the third year. As in other organs, the process may show itself by a solitary gumma or by a fibrotic condition of diffuse interstitial distribution. The epididymis not infrequently suffers during the secondary period, when, like the tertiary orchitis, it may be unilateral.

Nerve-lesions.—As already stated, the nervous system in tertiary syphilis is nearly as frequently involved as the skin and mucous membranes taken together. Nearly three-fourths of all spinal-cord diseases are of syphilitic origin. Under the heading of each cerebral or spinal disease the symptoms, pathological changes, &c., will be found described, so that only a passing notice is here required. Brain-syphilis is treated in detail in the article on 'Meningitis,' at page 1013. The lesions affecting the nervous system appear as a rule later than the skin and mucous-membrane lesions, but there are many notable exceptions. Fournier's figures show that in about one-ninth of all the cases of ordinary cerebral syphilis the symptoms of hemiplegia occur during the first year of the disease, and a much larger percentage of spinal cases occurs before the expiration of this early period. In a very small number of cases, sometimes described as 'malignant syphilis of the nervous system,' the implication of the spinal and cerebral centres has been observed before the disappearance of the induration of the primary sore. The term '*malignant syphilis*,' however, should be restricted to those cases of acquired syphilis in which the skin and mucous membranes are deeply

involved, the tertiary ulcerations supervening without any interval upon, or appearing along with, the secondary manifestations, leading to deep and widespread destruction of tissue and accompanied by grave constitutional debility, usually before the expiration of twelve months after inoculation.

The tertiary nerve-manifestations may be of the nature of gummata, which usually arise in the meninges of the brain or cord, or a specific meningitis, without gummatous growths, may occur; but more frequently these lesions arise from a true syphilitic arteritis, which involves the coats of the terminal arteries about the base of the brain. The gummatous growths may be solitary or multiple, giving rise to the signs and symptoms described under 'Cerebral Tumour,' in which headache, optic neuritis, hemiplegia, ocular paralysis, aphasia, convulsions, mental symptoms, and irregular forms of paralysis appear. In syphilitic arteritis many of these symptoms may be combined with others, due to secondary softening produced by thrombosis, and in all cases meningeal symptoms are common. The sclerotic nerve-changes, as seen in locomotor ataxia, chiefly affect the spinal system and belong to the parasyphilitic period, but in general paralysis sclerotic brain-changes may manifest themselves at a period as early as the third or fourth, though usually the advent is delayed till the seventh or tenth, year.

Insanity in any of its forms may be the only evidence of syphilis of the brain, all cerebral signs being absent, and there is reason for believing that the hereditary form of syphilis may also manifest itself in this manner.

Arterial Lesions.—Lesions in the arterial system, inside or outside the cranium, are invariably present, and constitute the most important pathological element in all cases of syphilis; endo-arteritis, panarteritis, peri-arteritis or endo-peri-arteritis all being constantly found sometimes in the same subject. The arterial changes may, in rare cases, manifest themselves before the end of the first year, especially in the neighbourhood of the base of the brain, but usually they are delayed till after the third year, and often do not give rise to symptoms till a much later period, when they may eventuate in atheroma of the aorta, aneurisms, or aortic regurgitation. Several types of syphilitic arteritis are recognised, the best-marked being the obliterative, in which the most advanced changes are seen in the inner coat of the vessels, constituting the condition known as *endarteritis obliterans*. In the *peri-arteritic* form gummatous nodules develop along the course of the small vessels; it is, however, common for this type to be found in the cerebral arteries, whilst the obliterating may be observed, if not in proximity, in some other part of the body. Spirochætae are constantly found in the altered vascular walls in the secondary stage, and sometimes in the arteritis

of the tertiary stage. The final results of the arterial changes are described under 'Arterio-sclerosis.'

Morbid Anatomy.—The histological examination of the primary sore or Hunterian chancre reveals the arterial changes described in the last paragraph. Not only are all the varieties of arteritis to be met with, but the veins and lymphatic vessels suffer in a similar manner. The histological structure of the tissue shows that the induration is the result of a characteristic small-celled infiltration, supported by a network formed through a hyperplasia of the cellular elements of the connective tissue. Large epithelioid cells and giant-cells are also present, and the spirochætæ, according to the reports of the most recent observers, are found in all parts of the lesion, as well as free in the narrowed lumen of the vessels. The histology of the chancre, according to Balzer, is identical with that of the papule in the cutaneous lesion characteristic of the secondary stage, though here there is, in addition to the obliterating and gummatous nodular peri-arteritis, a marked cellular infiltration of the cutaneous papillæ.

The histology of the gumma is that of an advanced developmental stage of the papule. The caseated cellular infiltration, consisting of small, large, epithelioid, and giant-cells, surrounded by the proliferation or hyperplasia of the connective-tissue elements, tends to soften, break down, and ulcerate in the centre. When resolution occurs, the cellular portion of the neoplasm disappears, its place being taken by dense fibrous tissue at the expense of the elastic fibres and of the normal constituents of the epidermic layer.

The gummata found in the viscera are of similar composition, the central caseating core being surrounded by a zone of fibrous proliferation, usually encapsuled by the tissue-elements of the viscus, into which the fibrous tissue of the gumma projects in the form of radiating processes.

SYPHILIS, CONGENITAL

Though there is practically little difference between the manifestations of the disease in this form of syphilis and in the acquired variety, save the absence of the primary chancre, nevertheless various important questions connected with its transmission arise.

Hereditary syphilis should be recognised as differing from congenital syphilis, though both forms are usually embraced under the latter title. Strictly speaking, the hereditary form of the disease is that which is handed down to the offspring through the spirochætæ in the seminal fluid of the father infecting the ovum of the mother. This is known as *sperm-inheritance*. The disease may be transmitted by a previously syphilitic mother to the offspring, through the

infected ovum, the father being healthy. This is known as *germ* or *ovular inheritance*, and is a more serious type of the disease than the former.

The term '*congenital syphilis*' is employed to designate the disease which is apparent in the infant at the time of its birth, or soon afterwards, exclusive only of those rare instances where an infant, healthy at birth, becomes infected by a syphilitic wet-nurse. This latter class of case obviously falls under the category of acquired syphilis.

The methods by which congenital syphilis is transmitted to the fœtus embrace the above-mentioned hereditary sperm and germ infections, the former occurring through the father and the latter through the mother. Obviously a third kind of inheritance may occur, when both father and mother are syphilitic at the time of conception; this is known as *mixed transmission*. Maternal transmission may, however, occur independent of these methods, when the mother becomes syphilitic *after* conception, the disease being in this case conveyed to the fœtus through the placenta; this form of conveyance is recognised as *placental transmission*, and is not likely to occur if the syphilis is acquired by the mother in the last six weeks of her pregnancy.

The infective nature of the semen of a syphilitic subject has been proved beyond question by its successful inoculation into apes. This important proof was necessary to the clinical evidences, since the remarkable fact was first pointed out by Colles of Dublin, that a healthy woman when she becomes impregnated by a syphilitic male conceives and brings forth a syphilitic child without herself manifesting any evidence of the disease. The infection of the fœtus in this case occurs through sperm-transmission, the father being free from local sores. Colles made the important observation, which is styled the '*Law of Colles*,' that such a healthy mother could with impunity suckle her syphilitic infant, though active specific ulceration of its mouth existed, as proved by the same infant infecting a healthy wet-nurse. The exceptions to this law are so few that they have been entirely denied by many authorities. It therefore follows that the mother is rendered immune by having conceived and brought forth a syphilitic infant. The important question arises as to how this immunity is accomplished, and it seems certain that the freedom from attack is conferred by her receiving from the fœtus an attenuated virus, its toxins or antitoxins. That she thus receives the disease in a latent form is undoubtedly proven in some cases by the advent in her person of tertiary or quaternary manifestations after the lapse of many years.

Conceptional Syphilis.—Sometimes, however, a previously healthy woman conceives after intercourse with a syphilitic male; the infected semen conveys the disease to the ovum,

and the syphilitic fœtus infects the mother, who may then show secondary manifestations during her pregnancy. Fournier offers a probable explanation of this rare phenomenon, and of the converse immunity in the great majority of instances ; he believes that the result depends upon the dose of the syphilitic virus received by the mother from the infected fœtus. When this is large, conceptional syphilis, with visible manifestations of secondary symptoms, occurs during pregnancy ; when the amount absorbed from the fœtus is small, Colles' immunity is established and may be permanent (*latent conceptional syphilis*), or the disease may show itself at a later period when tertiary symptoms supervene, in which case, according to Marshall, it should be regarded as delayed conceptional syphilis.

(These considerations are of the most vital importance as regards the possibility of the preparation of an immunising or therapeutic serum. Up to the present all attempts at such have proved futile, because it would appear that attenuation of the virus is practically impossible—it remains either actively capable of infecting or is totally dead.)

The existence of conceptional syphilis, as just described, is stoutly denied by several authorities, who maintain that when secondary symptoms occur in the mother during pregnancy these have followed a primary sore which has been overlooked, and that the disease is never conveyed in the roundabout manner of first reaching the ovum through the semen and from the ovum infecting the fœtus, from which it is again transmitted to the mother. That syphilis not very infrequently is transmitted to newly married women whose husbands have had no symptom of the disease for a couple of years is accounted for by Hutchinson and Fournier by the fact that the latter are liable to slight abrasions on the penis, called the 'after-marriage sore,' which may be infective ; and Hutchinson states that these abrasions are liable to appear about the site of the primary chancre.

Symptoms. —Syphilis communicated to the fœtus is liable to end in abortion, and it is a noteworthy fact that this result is much more likely to occur when the method of transmission is by maternal or germ inheritance. When the utero-gestation in these cases goes on to full term the infant may be born alive, but its tissues being so impregnated by the syphilitic virus, it often speedily perishes. More frequently it is expelled dead and often in a macerated condition during the later months of pregnancy, and the placenta and cord show diffuse vascular gummatous or other changes.

In the majority of cases of congenital syphilis, however, the child is born apparently healthy in appearance and often seems to be well nourished and even fat, the symptoms of syphilis developing within the first month or six weeks.

Infants born with all the manifestations of syphilis are prematurely aged in appearance, weak, wasted, and wrinkled. The skin may be covered with an eruption of a polymorphous type, on the wrists, palms, and soles pemphigoid bullæ are often visible, and bone-changes are obvious, sometimes in the form of well-marked epiphysitis; the liver and spleen are enlarged, and labial fissures or ulcerations are nearly always visible. Deformities are frequently present, and in one remarkable, and possibly unique, case delivered by the writer there were cleft palate, hare-lip, spina bifida, hydrocephalus, double club-foot, and cardiac malformations, the infant dying during the difficulty of delivering the enormously distended cranium.

In syphilitic infants born without evidence of the disease the first sign, according to Marshall, is often a loss of weight occurring within the first few weeks, notwithstanding normal feeding, and this may develop into a fatal marasmus before the appearance of any eruption. A diagnostic point of great value, according to this observer, is that the emaciation at this early stage is arrested by mercurial treatment.

Restlessness and whining, especially at night, usually precede the characteristic syphilitic rhinitis, with its sanious or purulent discharge. This latter sign is known by the name of 'snuffles,' owing to the noise which the infant makes in breathing and sucking, caused by the obstruction due to the thickened mucous membrane, which is frequently covered by crusts. The condition often extends along the Eustachian tubes, causing otitis media; and sometimes downwards, involving the larynx, producing laryngitis or laryngismus. The discharge from the nose leads to a characteristic fissuring of the upper lip, ulcerations soon appear about the angles of the mouth owing to the softening of mucous patches which have arisen upon the inside of the cheeks, and condylomata appear at the anus. The skin-eruption occurs about this period and is usually well developed by about the sixth week, rarely being delayed till the eighth or tenth. Erythematous, papular, but less frequently squamous or scaly syphilides appear, symmetrically distributed upon the buttocks, soles, palms, forehead, and around the mouth. Enlargement of the spleen is almost constantly present, and the liver often can be detected well below the costal arch. Iritis and choroid changes are also sometimes present, and the hair of the head and eyebrows is frequently shed. Sinking of the bridge of the nose, involvement of the cranial bones, or craniotabes, nodes, epiphysitis, dactylitis, and other manifestations resembling the ordinary tertiary lesions occur somewhat later; but as a general rule these, as pointed out by Hutchinson, are distributed symmetrically, forming a marked contrast to the tertiary lesions of the acquired variety.

Emaciation is almost constantly observed during the first six months of life or later, and the same remarkable physiognomy observed in those children born with advanced syphilitic signs becomes apparent: the child looks like an aged person—his face is withered or wizened. This appearance, popularly designated by the term 'old-fashioned,' is a condition which may long remain.

By the end of the first year the signs have usually passed away, and the child, though generally somewhat stunted, appears otherwise in good health. No further manifestations in many instances appear; but in some cases, especially when treatment has been neglected, relapses occur from time to time, and in a variable percentage of cases, somewhere about puberty or later (but occasionally as early as the second dentition), certain sequelæ appear, which may be regarded as in the main corresponding to the tertiary stage of acquired syphilis. These late manifestations or tertiaries are usually symmetrical; they consist of nodes on the tibiæ and forearms, serpiginous ulcerations affecting the skin or mucous membranes, keratitis, Parrot's bosses or bony elevations on the parietals developed from the early periostitis, deafness not the result of suppuration, and occasionally blindness from choroiditis. A most important series of signs are usually in evidence at a later stage of this period which are due to remnants of the infantile mischief and to arrest in the development of various organs or tissues. Since these latter signs are not infrequently present in adults in whom no history of congenital syphilis is obtainable owing to the disease not having been noticed, their recognition is of importance. Sometimes the arrest in development is general, so that the condition described by Fournier as *infantilism* is produced, the entire body being dwarfed. In most cases the skin is pasty and sallow or earthy in hue, the nose is depressed at its bridge or the lower part invaginated into the upper, constituting the so-called 'telescope' nose; this is, however, not due to an arrest in development, but to an early loss of substance in the nasal bones or septum. The forehead is square, the cranium often asymmetrical or natiform; the opacities caused by the remains of interstitial keratitis and the linear scars which radiate from the angles of the mouth all go to form a physiognomy which is hardly mistakable. Often the development of the testes, ovaries, and mammæ is retarded.

The Teeth in Congenital Syphilis.—Of perhaps more importance than any of these signs is the condition of the teeth, to which Hutchinson first directed attention and with which his name is usually associated. The milk-teeth in congenital syphilis are usually delayed in their advent through the gum, in company with other retardations in development,

as those of speech and locomotion ; and, moreover, they are prone to premature decay, but they present no characteristic malformations. It is only in the *permanent* set that an almost, if not quite, pathognomonic sign of congenital syphilis is to be sought for. This consists, as described by Jonathan Hutchinson, in a stunted growth of the teeth: 'they are narrow, rounded, and peg-like ; their edges are jagged and notched ; owing to their smallness their sides do not touch and interspaces are left.' Though all the teeth may show this dwarfed condition, it is the *central upper incisors* which suffer most and which often alone are malformed. The middle lobe of each of these is atrophied, which leaves a broad vertical notch in the free edge of the tooth as if a symmetrical piece had been filed out of each, 'and sometimes from this notch a shallow furrow passes upwards on both anterior and posterior surfaces nearly to the gum.' This characteristic notching should not be confounded with the condition in which the edges of the incisors are marked like the teeth of a saw, as this occurs in other diseases. The absence of Hutchinson's teeth does not, however, negative congenital syphilis, though its presence may be regarded as conclusive.

Many of the above signs, even including infantilism, may be present in the rare cases of syphilis acquired accidentally in infancy or early child-life ; but Hutchinson's notched teeth, the natiform skull, epiphysitis, involvement of the palms and soles, and the presence of snuffles are peculiar to congenital syphilis.

Rarely do quaternary manifestations, as juvenile tabes, appear ; but the degenerative effects or dystrophies and morbid predispositions are not rare, some of the victims developing epilepsy or insanity.

Pathological Anatomy.—The pathological changes of congenital syphilis in the main correspond with those of the acquired variety, and recent observers have as a rule found the spirochæta in all or most of the organs and lesions, so that the infectivity of the early stages of congenital syphilis constitutes a real danger never to be lost sight of. Moreover, the parasite has been found in the late lesions sometimes accompanying the congenital disease—a significant fact as regards the necessity for prolonged treatment.

Diagnosis of Syphilis.—In the acquired variety, before the appearance of the eruption the primary sore is liable to be mistaken for a *simple soft or non-syphilitic chancre*. The following points are to be observed: The true syphilitic chancre appears first after an incubation-period dating from contact till about the twenty-fifth day—viz. between three and four weeks. The soft chancre can hardly be said to have an incubation-period, since it usually shows itself within a day or two after exposure ; but the syphilitic virus may be

introduced at the same time, in which case a true chancre will develop upon the same or some adjoining site after the soft sore has disappeared. In other cases a patient with an overlooked syphilitic papule may in a later intercourse have this infected by a soft sore. These *mixed* chancres may be most puzzling, and a diagnosis by the length of time of the first incubation-period will be impossible when promiscuous intercourses have been indulged in. The soft sore is usually multiple, the true one rarely so; the edges of the former are clear-cut and seem as if punched out. It is from the first obviously an ulcer surrounded by a margin of redness, secretes pus, has a soft irregular base with a yellowish centre, and is often accompanied by an acute inflammatory bubo. The specific strepto-bacillus of Ducrey can be detected in the pus, and a very important character of the soft sore is that it is auto-inoculable: a drop of the pus inoculated into the healthy skin in the vicinity of the sore will produce a typical soft sore. These characteristics are all in marked contrast to the true sore, which, when pure, never strictly ulcerates; appearing as a simple erosion of a grey colour in the centre, it has no punched-out or undermined edges, and discharges but a slightly red-stained moisture, and by the end of a week exhibits an unmistakable cartilaginous hardness which is highly characteristic. The bubo in the groins is also markedly different in each case; the hard shotty feel of the small, painless, indolent tumour which always accompanies the true sore is in marked contrast with the acutely inflamed glands which in the case of the soft sore form when the latter becomes inflamed.

Notwithstanding the above-mentioned distinctions, it is usually unwise to pronounce any sore to be non-syphilitic till after the expiration of the total incubation-period; this is especially important, since in some very rare cases the true sore has been known to occur without induration developing. If the eruption has already appeared when the patient presents himself for examination, usually there is little difficulty in the diagnosis. The coppery tint and polymorphism are at once suggestive; there is usually a copious amount of the eruption symmetrically distributed first upon the abdomen and chest, afterwards spreading to the extremities and often to the face, avoiding the backs of the elbows and fronts of the knees. At one place it may be roseolar, as on the belly, whilst upon the forehead it may be so markedly papular as to simulate acne; in other regions it may be squamous, pustular, or, more rarely, pemphigoid.

The rash usually comes out in crops, a papular often following a roseolar form, in which case the latter remains usually mixed with the papules. There is commonly slight fever present, and in the rare cases where this is marked the case

may simulate mild *scarlatina* or *measles*, but the condition of the mucous membranes and the discovery of patches in the mouth and snail-track ulceration on the tonsils leave no doubt.

Lichen planus is differentiated from the papular syphilide by the smooth, shining, flat character of its papules, which coalesce into characteristic groups, are accompanied by itching, and leave pigmented staining; whilst the small papules of syphilis are markedly convex and hard, often remaining discrete, and are not accompanied by itching. There is no other skin-condition in which it is so absolutely necessary to inspect the entire cutaneous surface as in the skin-syphilides: the gyrate outlines, the symmetrical distribution, and the polymorphism of the eruption only become evident on a complete survey.

Palmar eczema may be differentiated from a squamous syphilide by its not being strictly limited to the palmar surface; it nearly always creeps round the inner margin and involves the dorsal aspect more or less, after having attacked the entire palms on *both sides* of the body. There is no thickening or induration of the underlying tissues; the margins of the eruption are never serpiginous, but fade into the surrounding healthy skin; in all of which features it differs from the squamous syphilide.

Lupus may be mistaken for the ulcerative syphilide, but the presence of the apple-jelly nodules is pathognomonic; when these cannot be detected, the history and slow progress of the lupoid ulceration, which is superficial, with its delicate undermined but not infiltrated irregular margins, are usually characteristic.

Diphtheria may be simulated by the mucous patches on the tonsils, but the absence of the Klebs-Loeffler bacillus and the other signs of syphilis always present should prevent mistakes. The different forms of *stomatitis* are as a rule easily differentiated, save one—*mercurial stomatitis*, since this is so frequently present as a complication in syphilitic patients taking mercury. The question which presents itself to the physician in such cases is how much of the mouth-condition is due to the drug and how much to the spirochæta; the presence of fœtor and salivation, the localisation of the ulcerative lesion starting on the spongy gum about the last two molars, point decidedly towards mercurial stomatitis. (See under 'Stomatitis.')

In the tertiary stage of the disease the cellular and periosteal nodes may be mistaken for *malignant growths*, but the circumscribed, indolent, and painless features of the cellular node, which in its early stages never is adherent to the skin, are sufficient to distinguish it from a cancerous neoplasm; which, moreover, always involves the neighbouring lymphatic

glands, whilst in the tertiary syphilides these escape almost invariably. The nocturnal pains constantly associated with periosteal nodes are very characteristic of syphilis.

A cutaneous gumma on the leg after suppuration is often mistaken for a *Varicose ulcer*; but the latter always occurs singly, usually a little above the ankle, has thickened callous edges, surrounded usually with scaly eczema, contrasting markedly with the syphilitic ulcer, whose site is always higher up near the knee, has punched-out edges, a greenish-yellow caseous core, is circular in outline, and often multiple.

The difficulty is greater in the differentiation of an ulcerative gumma of the tongue from *Cancer of the tongue*. The gummatous ulcer is as a rule much less painful than the malignant, and there is very seldom marked lymphatic gland-involvement; its edges are clean-cut, and the central yellow wash-leather core is highly characteristic. The constitutional symptoms of tertiary gummatous glossitis are never so grave as in malignant disease, unless the deep ulceration causes such pain in the movements of the organ as interferes with mastication and deglutition; but severe spontaneous pain when the tongue is at rest is usually entirely absent, and the condition readily yields to iodides as a rule. *Leucoplakia* of the tongue is most certainly in the great majority of cases of syphilitic origin, often appearing in the quaternary stage; hence it is not amenable to either mercury or iodides, and in a large percentage of cases malignant disease of the epithelial type finally supervenes upon the leucoplastic patch. The history of the slow evolution of the hyperkeratosis of the pearly-white raised patch of the epithelial layer of the tongue usually leaves no doubt in the diagnosis.

Nerve-lesions in tertiary syphilis are so numerous, and their symptoms so varied, that their diagnosis cannot be here discussed; moreover, the subject is treated under the head of each affection, as 'Cerebral Tumour,' 'Meningitis,' 'Myelitis,' 'Paralysis of the Ocular Nerves,' &c. Hutchinson points out that when paralysis involves only one nerve-trunk, especially a cerebral nerve, syphilis is probably the cause, and he believes that fully 50 per cent. of all cases in which paralysis affects the third, fourth, fifth, and sixth nerves, when only one of these is involved, is syphilitic and curable. The third-nerve paralyses are recognised to be notoriously syphilitic, and they may be the first and sometimes the only sign of cerebral syphilis.

In the investigation of the syphilitic origin of a nervous lesion often the only data available are the scars of the former struggle, as the adhesions left by an old iritis, or the cicatrices of past serpiginous ulceration, since in a not inconsiderable percentage of cases there is no history of the primary or secondary stage, the facts being either voluntarily suppressed

or being unknown to the sufferer. This latter state of affairs occurs oftenest in married women. In these the history of repeated miscarriages should always arouse suspicion. In many cases the diagnosis is only cleared up by the results of treatment, the tertiary manifestations yielding with readiness to iodides as a rule.

The diagnosis of congenital syphilis has been already indirectly dealt with. One important point arising in connection with the diagnosis of congenital or heredo-syphilis has not, however, been referred to. This is the question which has been so much disputed as to whether syphilis can be transmitted to the third generation. Of late years the evidence in favour of such an event has been steadily accumulating, and it is no longer seriously doubted by most syphilologists that a syphilitic grandparent may hand down genuinely active syphilis to his grandchild. The evidence (from which obviously re-infections must be rigidly excluded) shows that stigmata, as Hutchinson's teeth, infantilism, cranial malformations, keratitis, &c., are frequently observed in the third generation, and that active lesions are by no means so uncommon as have been supposed. E. Fournier has published and collected cases in which the disease appeared even in a *virulent* form in the third generation. These cases will be found in Marshall's excellent volume on 'Syphilology,' and this observer, whilst acknowledging the possibility of transmission of active syphilis to the third generation, points out the difficulty of excluding a possible re-infection and the possible intervention of another syphilitic genitor.

That the degenerative or dystrophic effects of syphilis are transmitted to the third generation has been always accepted, but this is obviously a different question from the transmission of active syphilis. The spirochæta has been found in the ovaries of patients succumbing to active congenital syphilis, and its infectivity has been demonstrated by the successful inoculation of apes. These facts, taken in conjunction with the successful production by Neisser of a chancre in an ape by the inoculation from a gumma in the seventeenth year of syphilis, afford reason for the belief that the actual demonstration of transmission to the third generation will soon be placed beyond question. Marriages before the seventeenth year amongst subjects one or other of whom has suffered from congenital syphilis are not rare, and suspicious symptoms occurring in the offspring of such marriages should be duly weighed in the light of the more recent bacteriological advances made in the elucidation of syphilis.

Prognosis.—The outlook is so much a question of the thoroughness and duration of treatment that it need hardly be discussed. Few sufferers from syphilis die, except from remote effects, when proper treatment is commenced early ;

and a good prognosis in a given case largely depends upon the absence of any previous disease which prevents mercurialisation of the patient. Malignant syphilis—*i.e.* that type in which tertiary ulcerations appear in the ordinary secondary period—has a very grave prognosis, since these patients almost invariably bear mercury badly.

Alcoholism always intensifies the severity of the disease. As a rule, no assistance is obtained by a study of the primary sore; the most severe tertiary manifestations often follow an insignificant papule. The statement is made that the severity of the attack is in proportion to the amount of induration seen in the primary lesion; this cannot be accepted as a general rule, profound infection having been known to follow a sore which never showed signs of induration. Phagedænic ulceration setting in during the primary stage usually is followed by a very severe type of secondary ulceration and often by grave early involvement of the nervous system. Whether this latter type of syphilis, which is styled by Giles de la Fourette as 'Early malignant syphilis of the nervous system,' be ushered in with phagedæna or not, it has always a grave forecast.

A very important subject in the prognosis of syphilis, whether first seen in the primary or secondary stage, is the question of the degree of the future liability to tertiary and quaternary manifestations. The answer admits of little doubt in the majority of cases, and it is clearly the duty of the physician to lay before the patient at his first interview the importance of the fact that the final results of his attack depend largely upon himself. If he rigidly perseveres in a prolonged mercurial treatment the liability to late lesions is reduced to a small minimum; according to Fournier, not more than one patient in twenty suffers from tertiaries when properly treated for a period of at least three years' duration.

Treatment.—Mercury is the only drug of real value in the treatment of the earlier stages of syphilis, its curative action being as obviously demonstrable as that of iodides in the tertiary period. How the discovery of this remarkable specific action of mercury was effected is unknown, and the subject is one of the most remarkable in the whole range of therapeutic empiricism. The treatment of the different stages of syphilis, with their endless manifestations, complications, and sequelæ, is a wide subject, which the writer has discussed in detail in his 'Dictionary of Treatment' (4th ed.), pp. 916-930, and only general principles can be here referred to.

The management of the primary chancre admits of little dispute; cauterisation, excision, &c., are absolutely useless once the syphilitic sore has formed. Black-wash is one of the best routine local applications, applied to the sore on lint. Little need be said of Prophylaxis, and this little may be

summed up in the single word *purity*. Should a *prophylactic* serum be discovered, it is probable that the ultimate consequences to the human race might be more serious than even the ravages of the disease, since the total removal of the barrier—the dread of catching syphilis in illicit intercourse—would lead in the long run to evils of a worse type. The question of whether anything can be done to prevent syphilis developing after contact or inoculation—say, when the surgeon or accoucheur has wounded his finger in an operation or autopsy on a syphilitic subject—is a matter of considerable importance, independent of all moral considerations. This has been practically solved by the recent experiments of Metchnikoff, who demonstrated that a 30 per cent. calomel-ointment rubbed into the site of a syphilitic inoculation within an hour after the virus prevented the formation of a primary sore on the glans penis of a medical student and of a macacus monkey.

As regards the advisability of commencing constitutional mercurial treatment as soon as the primary sore is discovered there is still considerable differences of opinion, though it can be no longer doubted that early active treatment at this stage, as shown by Hutchinson, may entirely prevent the advent of secondary symptoms and render the appearance of tertiary manifestations a remote possibility. The only objection to this method is one which works out as a serious difficulty with many patients and even with some physicians, because when the evidences of constitutional infection are by this plan prevented or rendered latent there must always remain a doubt about the specific nature of the primary sore. This doubt is fatal to whole-hearted persevering mercurial treatment. Hence the writer's routine plan is to commence with *small* doses of the drug immediately after the appearance of any sore warranting a probably correct diagnosis of syphilis, but not to push it to the extent of quite aborting the secondary symptoms. Upon the first faint but unmistakable sign of a syphilitic roseola or mucous patch, the patient and physician being absolutely convinced of the correctness of the diagnosis, the dose is immediately increased or thorough inunction is started, which must be continuously kept up for a period of at least nine months, and afterwards, with interruptions or intermittent periods, for a full period of three or four years dating from the appearance of the sore. The preliminary mild mercurial course undoubtedly, when skilfully carried out, modifies the disease and renders the subsequent vigorous treatment more telling. Five grains of grey powder in divided doses spread over the twenty-four hours may be taken as a fair average, but the physician should refuse to permit himself to be tied down by any preconceived idea of dosage. The drug in its various combinations or in the free

state, whether given by the mouth, rubbed into the skin, injected into the subcutaneous tissue, or otherwise employed, should be pushed till evidence of safe saturation is produced, salivation being carefully avoided; the dose may then be often reduced, but, if possible, not suspended. Large dosage too often affects the mouth somewhat suddenly, so that suspension must be resorted to; hence the most careful attention to the condition of the gums is essential from day to day, if possible, till the patient's capabilities of bearing the metal are demonstrated.

In the article already referred to the writer insists upon the value obtainable by weighing the patient at regular short intervals; as a rule, as long as the body-weight increases or keeps stationary there is little danger in pressing the drug.

Much divergence of opinion still exists about the relative merits of the so-called *continuous* and *intermittent* plans of administering mercury in the cure of syphilis, and unfortunately confusion arises from failure in recognising the meaning which different syphilologists attach to these terms. The writer has no doubt about the wisdom of adhering to the continuous method during the first nine months, and commencing the intermittent plan later on. Fournier, with whose name is associated the method known as the *chronic intermittent* plan, believes that during the intermissions, the mercury being eliminated entirely from the system, the virus becomes more susceptible to its influence when the administration is recommenced. This authority consequently for the first two years advises treatment to be pursued, for periods of six weeks at a time, separated by similar or shorter periods of rest, for two whole years, when the mercurial administration is suspended entirely, during the third and fourth year to be commenced again, and to be continued in an intermittent manner during the fifth, seventh, or eighth year, making in all a total period of four years' duration, in which treatment is pursued in an intermittent fashion. There can be no doubt but this method is incomparably better than a routine year of active continuous treatment and a permanent cessation from hostilities, which is almost certain to lead to renewed outbreaks later on. One great advantage of the many which the discovery of the *spirochæta pallida* has already brought is the conviction that a longer period of treatment by mercury is necessary, since the parasite has been demonstrated in an active form in some old tertiary lesions. It is probable that the ideal mercurial treatment in the future will be considered to be a more continuous type of administration during the first nine months, with frequent interrupted periods of administration, extending over a period of several years, without the long rest of two years at the end of the second year's treatment, as practised by Fournier.

Iodides are employed by many in the secondary stage of syphilis, but as a rule iodine in any form should not be given during the first six months. It has a remarkable action over the neoplasms of the tertiary period, but, as maintained by Fournier and Marshall, the drug has no curative power over syphilis in the sense that mercury has; though it dissipates the gummatous lesions with certainty, it has no preventive influence over the future course of the syphilitic process. The tertiary manifestations, as gummata, nodes, serpiginous ulcerations, visceral and nerve-lesions, disappear under the influence of the drug as if by magic. To produce this result, however, very large doses are often, if not always, necessary; nothing is more common than to see a sternal or clavicular node resist for many months a 5 gr. dose of iodide of sodium or potassium, when rapid dissipation or dispersion of the gumma follows, sometimes within a week, after the dose has been increased to 20 or 30 grs. three or four times a day. In 10 gr. doses it may be given as a routine during the second year, alone in the intermissions of mercurial treatment, or in combination with the perchloride of mercury when the mercurial treatment is resumed.

In all cases of tertiary syphilis the iodide-treatment should be followed by a longer or shorter course of mercury, and very frequently arsenic proves a valuable adjunct. It will be found upon the whole better in the treatment of tertiary gummata to give the iodide alone, especially when the symptoms are urgent, as in brain-syphilis, unless previous mercurial treatment has been neglected. When the neoplasm shows signs of resolution the dose of iodide may be reduced, and mercury given in combination with it. In the late secondary period, where the manifestations of the disease, especially the mucous-membrane and skin lesions show a tendency towards the tertiary type, the combination of mercury and iodides should be freely pushed. In these cases, however, it is very necessary to remember that iodides frequently cause a severe eruption which may be mistaken for a recurrent syphilitic rash.

The relative merits of the different mercurial preparations, and of the different methods of administering the drug, by the mouth, hypodermically, by inunction, fumigation, &c., are discussed in the author's article already referred to. Suffice it to say that the inunction plan is the most rapid and effective of all methods save one, the exception being the new intravenous injections of the cyanide or biniodide of mercury which are associated with the name of Bacelli. This plan is justified in malignant syphilis, and especially in early malignant syphilis of the nervous system.

The treatment of mercurial stomatitis, as well as the symptoms, signs, and diagnosis of the condition, are discussed in the article on 'Stomatitis.'

Congenital Syphilis.—The treatment of hereditary and congenital syphilis must be conducted upon the same lines as that of the acquired variety, but important matters arise in this connection regarding preventive measures.

Prophylaxis.—The first question to be settled is the serious one: How soon should a syphilitic patient be permitted to marry? Hutchinson lays it down as a rule that marriage in a male may be permitted after *two years* of treatment dating from the primary stage, but that a longer period must be insisted upon in the case of a woman. The great majority of syphilologists, however, insist rightly upon a period of *at least four years* in all cases. It appears almost certain that the two years' period will be accepted as unsafe as soon as the recent advances in the bacteriology of the disease are duly recognised and realised. Even after the lapse of four years from the primary sore, no matter how thoroughly the treatment has been carried out, the writer's invariable plan is to insist upon a short mercurial course of, say, four to six months immediately before marriage. After conception, mild mercurial treatment of the female should be carried out, when this can be effected without arousing a suspicion which may embitter the lives of both parties. In premature marriages, when the husband is found to be suffering from later secondary or early tertiary manifestations, the pregnant female should be always submitted to active treatment by mercury. By this means healthy children are usually born to syphilitic parents, and the efficacy of mild mercurial treatment of the mother is constantly demonstrated by the birth of a non-syphilitic infant, after one or more miscarriages had previously occurred, when no mercury had been taken. As soon as the first signs of syphilis appear in an infant after birth mercury should be administered without hesitation, even in the presence of obvious marasmus. The antagonism between the metal and the syphilitic virus is demonstrated in the remarkable improvement which speedily occurs in the general nutrition, as well as in the disappearance of the eruption. Preconceived notions of the toxic effects of mercury upon the healthy organism are liable to paralyse the physician in the treatment of the congenital malady. Once the idea is firmly grasped that the active toxins of syphilis antagonise the deleterious effects of the metal, as pointed out by Hutchinson, the therapist begins to feel that he is treading upon safe ground. Here, even more obviously than in the acquired variety of the disease, the value of repeated and systematic observations upon body-weight become demonstrated.

In the author's 'Materia Medica' the treatment of congenital syphilis is summed up in the following paragraph, written many years ago, which contains all that need be here

said on the subject: 'In congenital syphilis there is no preparation equal to grey powder, which may be given freely, as mercury in moderate doses seems almost incapable of doing harm, whilst there is a large amount of the syphilitic poison for it to expend itself upon. Weak emaciated infants bear larger doses when poisoned with syphilis than they can when afterwards apparently cured and fattened; but if after a period of neglect syphilitic symptoms come on in a marked form, then they bear very large doses again. A child half a year old may get $\frac{1}{2}$ gr. of grey powder three times a day for three days, then $\frac{1}{2}$ gr. every night, and this may be continued as long as the infant thrives. If no result seems to follow, a little of the ointment of mercury may be rubbed in occasionally.' The only change which subsequent experience has dictated in the above statement is that the dose should be increased as the child grows older. The discovery of the spirochæta in the late manifestations of congenital syphilis proves the absolute necessity of continuing mercurial treatment for a period quite as long, if not longer, than that indicated in the acquired form—viz. for at least four or five years.

Both mercury and iodides appear to possess no appreciable action over the parasyphilitic or quaternary manifestations of the disease; at this advanced period the typical syphilitic granulomatous growth and the spirochæta have disappeared.

SYRINGOMYELIA

This is the term applied to a slowly progressive disease of the spinal cord, in which one or more cavities exist, the result of developmental anomalies, associated with a central gliosis or proliferation of the embryonic or adult neuroglia-tissue in the neighbourhood of the central canal.

Pathological Anatomy.—In the condition of the cord known as hydromyelia there is dilatation of the central spinal canal, which is surrounded by a layer of embryonic cells. When these cells remain permanently in their foetal condition the abnormality gives rise to no clinical symptoms. In syringomyelia there is obvious proliferation, either of this embryonic zone or sheath, or of the normal adult gliomatous tissue which surrounds the central canal. The tendency towards progressive degenerative cavity-formation in this new gliomatous neoplasm constitutes syringomyelia. The cord in most instances exhibits one large canal or cavity, which may occupy a considerable extent of its length, the new cavity having the central spinal canal situated in front of it, and often in communication with it. On transverse section it may be seen to occupy a large extent of the entire

thickness of the wasted cord, or it may appear in some sections as a small circular or stellate aperture or fissure, situated more or less centrally. Though this new canal may extend from the medulla to the termination of the cord, it is more common to find it limited to the dorsal or lower cervical region, and sometimes several cavities co-exist. Its contents consist of clear or opaline fluid; a layer of epithelial cells lines its interior surface, outside of which is the sheath of translucent gelatinous, embryonic, gliomatous tissue, consisting of spindle-cells and fine fibres forming the neuroglial network. This new proliferated tissue, even when not cavernous, may appear in parts of the cord as small tumours, causing considerable enlargement or flattening, and not infrequently degenerative system-lesions are visible in the white columns. Dilatation of the Sylvian aqueduct and internal hydrocephalus, causing flattening of the cerebral convolutions, are sometimes observed in young subjects.

Etiology.—The condition is found much more frequently in males than in females, most commonly exhibiting its presence sometime between the twentieth and thirtieth years. Trauma and syphilis have been suspected as etiological factors, but the frequent combination of various obvious developmental anomalies, as extra digits, lop-sided craniums, spina bifida, &c., in patients suffering from syringomyelia, as well as the presence of the pathological changes above described, have led to the general acceptance of the view that the condition is due to some error in development which only manifests its presence in a proportion of the cases, and this, too, at a period long after birth, when the embryonic gliomatous tissue begins to proliferate.

Symptoms.—These vary greatly with the regions and with the elements of the cord affected, and, as just stated, they may be entirely absent, the condition being unsuspected, and only revealed at an examination after death from some other disease.

The most characteristic and constant, and often the first noticeable, clinical feature of syringomyelia is a remarkable disorder of sensation known as *dissociation* or, as more correctly styled, *dissociated anæsthesia*, in which, whilst the tactile and muscular senses are normal, sensations of pain and temperature are either markedly diminished or absent entirely. This symptom is manifested by the frequency with which patients unconsciously permit themselves to be burned, scalded, or otherwise injured. The areas of the skin so affected are seldom symmetrical, and are often confined to the upper extremities, chest, and face, the legs being only involved during the later stages of the gliosis. Many peculiarities are observable in the same or in different cases; thus, the sensation may remain for cold, whilst that for heat is

lost, or *vice versa*; and very occasionally the tactile sense in some areas may be impaired. The explanation of dissociation is found in the theory that thermal and painful impressions are conveyed by fibres which pass at once deeply into the grey matter and decussate in the posterior commissure, whilst those conveying tactile impressions remain less centrally situated in the postero-lateral columns which escape. Muscular atrophy, affecting chiefly the upper extremities, is common; it is of the usual type seen in the progressive form of muscular wasting, commencing in the thumb-muscles and invading those of the fore-arm, arm, and shoulder, and frequently producing the griffin-claw deformity. The legs are ultimately involved, the first sign being usually an increase in the knee-jerks, followed by gradual loss of power, and rigidity, forming a clinical picture of spastic paraplegia or amyotrophic lateral sclerosis. Scoliosis, lordosis, kyphosis, and deformity of the thorax may develop.

Symptoms arising from interference with the origin of the bulbar nerves on one or both sides are also but very rarely seen; paralysis of the sympathetic in the cervical region is sometimes to be observed, causing contraction of one or both pupils. Vaso-motor disturbances, causing lividity of the fingers, and trophic changes in the skin, bones, and in the ends of the digits and nails, are common, but a much more important series of trophic changes are those which involve the joints, and which occur in nearly a quarter of the entire cases of the disease. The shoulder and elbow joints, according to Schlesinger, are by far the most frequently involved, in contrast to the apparently identical arthropathy which occurs in locomotor ataxia, and which, in the great majority of cases in this latter disease, destroys the hip and knee joints. Pains in the back and in the arms, often associated with tingling and formication, are commonly present, but are rarely severe. The diversity of the symptoms of syringomyelia has led to the division into distinct types, but these eventually may run into each other as the gliosis advances; thus, the authority just mentioned recognises from the regional point of view a cervical, dorso-lumbar, sacro-lumbar, and bulbo-medullary type. Edwin Bramwell rightly insists that it is in the *character* of the symptoms rather than in their *distribution* that their main diagnostic importance lies, and hence several types are recognised, founded upon the characters of the symptoms independently of their distribution; thus, there is recognised a *motor* form of syringomyelia, in which the clinical picture is that of a typical amyotrophic lateral sclerosis in which the dissociation may be for a long time absent, or only be discovered with difficulty in some small area of the skin. An *atrophic* type may be met with, practically identical with progressive muscular atrophy, from which

affection it can only be differentiated by the presence of a small patch of dissociated anæsthesia or by the existence of some trophic change. The *tabetic* type is a form of the disease described by Oppenheim, which closely simulates locomotor ataxia, but this type is usually associated with the distinct classical symptoms of the common cervico-dorsal variety in the upper extremities. An *hysterical* type is described in which atrophic and trophic signs may be long entirely overshadowed by the marked sensory symptoms of dissociated anæsthesia combined with symptoms resembling functional hemiplegia or hemianæsthesia. The *trophic* type of syringomyelia is constituted by the predominance of the trophic changes, and was at first regarded as a distinct entity under the title of Morvan's Disease. The chief signs in this form of the disease are *numerous painless onychias* or *destructive whitlows*, usually associated with severe neuritic or neuralgic pains in the arms, joint-changes in other regions, spontaneous fractures, often wasting of the upper extremities, and analgesia or anæsthesia of the skin covering them.

The progress of the disease in all its forms is usually a slow one, extending often over many years, during which marked ameliorations occur in the patient's condition, indicative of a halt in the usually progressive nature of the gliosis. The special senses generally remain unaffected throughout, and the sphincters are only liable to suffer in the lumbo-sacral type of the disease.

Diagnosis.—The chief points in the elimination of *spastic paraplegia*, *amyotrophic lateral sclerosis*, and *progressive muscular atrophy* have already been incidentally emphasised in the description of the types of syringomyelia. The essential points in the diagnosis of the disease are the presence of trophic changes, as onychia, Charcot's joints, and the dissociated anæsthesia, when taken together with symptoms of muscular wasting of the Duchenne-Aran type in the upper extremities, or of amyotrophic lateral sclerosis. From this latter disease syringomyelia is further differentiated by the absence of the increased biceps and triceps tendon-jerks, of the jaw-jerk, of fibrillary twitchings and abnormal excitability of the muscles to direct mechanical stimuli; but when amyotrophic lateral sclerosis extends to the bulb the bulbar type of syringomyelia is simulated. Bulbar syringomyelia is characteristic not only in being associated with the typical trophic and sensory dissociation, but it runs usually a much slower and more chronic course than other forms of bulbar degeneration or disease.

Disseminated sclerosis is discriminated by the scanning speech, nystagmus, optic-disc atrophy, vertigo, intention-tremors, &c., and is eliminated by the absence of dissociated anæsthesia and trophic changes when spasticity and increased knee-jerks have developed.

Hysteria can only be eliminated by the demonstration of symptoms arising from lesions of an organic nature, and the search for trophic changes should be thorough; actual wasting, except such as follows from long disuse, never occurs in hysteria. The remarkable sensory disturbances met with in hysteria may simulate dissociated anæsthesia; but though the sensation of heat and of cold and of pain may be entirely lost, the tactile sensibility also suffers, and the distribution (hemianæsthesia), combined with hyperæsthesia of the special senses, is suggestive. The symptoms of hysteria may appear in a patient suffering from unmistakable syringomyelia, as they occasionally accompany nearly every other form of organic nerve-lesion in hysterical subjects.

Cervical pachymeningitis runs a rapid course, but the preponderance of the sensory symptoms—viz. the very severe shooting neuralgic pain down the arms—is characteristic. There is wasting of the upper extremities, but the anæsthesia which is present involves all the varieties of sensation and never appears in the typical form of dissociated anæsthesia, and there are no trophic changes present, unless in the rare condition in which both diseases occur together.

Tumours of the spinal cord may produce typical dissociated anæsthesia, but the history of the case and its rapid progress, especially if associated with Brown-Séquard's paralysis, will aid in the diagnosis; pain is, moreover, usually a more marked symptom than in syringomyelia. When the tumour is central in site the symptoms may be as symmetrical as in syringomyelia, so that the differential diagnosis can only be guessed at, and, as pointed out by Gowers, such growths generally arise from residual gliomatous tissue and are therefore associated with syringomyelia. It is only by the more rapid development of the symptoms and their more speedy extension that the nature of these cases can be surmised.

Hæmorrhage into the cord, or acute central hæmorrhagic myelitis, should, as pointed out by Edwin Bramwell, always suggest a previous condition of syringomyelia, which may be verified by a scrutiny of the history of the case. Hæmorrhages are common during the progress of the gliosis, and it is not rare to find blood present in the cavity, even when no acute exacerbation in the progress of the case had been noticed prior to death.

Anæsthetic leprosy, with its resulting neuritis, is closely simulated in some cases, since in that disease dissociated anæsthesia has been occasionally observed; trophic changes and muscular atrophy also occur. The history, the progress of the case, and the discovery of the leprosy bacillus in an excised piece of nerve usually settle the diagnosis when nodosities upon the superficial nerve-trunks cannot be detected.

Prognosis.—The condition invariably progresses towards a fatal issue, but owing to its slow course over many years, and its not infrequent periods of inactivity, death frequently occurs from some intercurrent affection. The prognosis is gravest in those cases where the lumbo-sacral region of the cord is most seriously or alone involved, as the bladder is affected at an early stage in this variety of the affection.

Treatment.—No known method of treatment has any influence over the progress of the malady, and the only course which can be pursued in the management of the case is such as is indicated for the relief of symptoms, the avoidance of traumata, and the prevention of sepsis from the urinary tract or from the sites of trophic lesions.

TABES DORSALIS. (See under ATAXIA, p. 87.)

TACHYCARDIA. (See under HEART, p. 564.)

TAPEWORMS, or TÆNIÆ

These parasites belong to the family Cestoda; they are of a flat or ribbon-shaped form, in which many distinct and sexually mature individuals, recognised as proglottides or so-called 'joints,' are connected in one colony or body known in zoological language as a strobila. The colony is provided with a head, armed with hooklets or suckers, or both. Immediately succeeding the head is a slender neck of homogeneous structure which, in its lower portion, is seen to be composed of rudimentary proglottides; these, as they are observed further down the colony, become larger, till at a variable distance from the head and neck (which is called the scolcx) each proglottis is found to be sexually mature, containing male and female genital organs, with the female organs distended by fully developed ova in each proglottis near to the end of the ribbon-shaped mass. The parasite anchors itself, by means of the suckers and hooklets arranged round its rostellum, to the mucous membrane of the small intestine, and the entire colony nourishes itself by absorbing through each proglottis the intestinal juices of its host. A complicated water-vascular system of canals extends throughout the length of the strobila.

A study of the life-history of the parasite shows that it requires an intermediate host. The last joints or proglottides of the fully developed and mature parasite are shed, and

pass out with their contained ripe ova in the fæces. The segments having disintegrated, the embryos are set free, unless the fresh proglottis has been swallowed by its intermediate host soon after its expulsion from the human body. Probably the ova are usually first freed, and find their way into water or amongst the stems of vegetables, aquatic plants, grasses, &c., before reaching the stomach. In the most frequently met-with tapeworm, known as *tænia solium*, the ova are swallowed by the pig. Having entered the stomach of this animal, the shell of the ovum is digested, and by the six hooklets with which the embryo is armed it works its way through the layers of the intestinal tube, enters the blood-stream, and, finally, takes up its abode in the connective tissue between the bundles of the muscular fibres or in the interstices of any of the viscera. In this stage of its existence the parasite is a small bladder-like cyst, known as *Cysticercus cellulosæ*, constituting the disease known as 'measles' in the pig.

The cyst next develops a head and neck, or scolex, resembling that of the mature tapeworm, which it is capable of protruding or withdrawing into its interior. This bladder-stage corresponds to the hydatid stage of a small tapeworm of the dog which produces the disease known in man as hydatids, described upon page 647. In this phase of its existence the *cysticercus* remains for an indefinite time, or perishes, unless the meat of the infested pig be eaten raw or in an imperfectly cooked state by man, in which case the cystic part of the organism is destroyed by the human gastric juice, and the scolex, by means of its suckers and teeth, moors itself to the mucous membrane of the intestine, and, producing a long series of proglottides, becomes developed into the mature parasite or strobila known as *tænia solium*, which lies free or coiled up upon itself in the small intestine. Whilst this cycle may be taken as in the main typical of the development of tapeworms in general, in the bothriocephaloid family no cystic or bladder stage is developed in the tissues of the intermediate host, which is some species of fresh-water fish, the larvæ of the parasite retaining for many years a worm-like form till eaten by man or some other final host.

Many specimens of *tænia* have been from time to time found in man, but three only need be considered as of any practical clinical importance—i.e. *Tænia solium*, *Tænia mediocanellata*, and *Bothriocephalus latus*.

TÆNIA SOLIUM.—This parasite is known as the pork-tapeworm, whose life-cycle has been just described. Its title 'solium' originated in the mistaken belief that it only occurred singly in man, but it is now recognised that two, three, or more may exist together in the human intestine. It is only derived from eating raw or imperfectly cooked

pork, bacon, or ham; hence its geographical distribution in man corresponds with that of the pig or with the commercial distribution of its flesh, and it is especially common in Germany, where smoked ham is often eaten without being cooked.

The anatomical peculiarities of this species should be recognised by the practical physician, since effective treatment can often only be carried out when a correct diagnosis is arrived at, as the head is dislodged with considerable difficulty owing to its duplex armature of suckers and hooklets. The total length of very large specimens rarely exceeds twelve feet, more frequently not measuring beyond six or eight feet, being thus considerably shorter than the meat and fish tapeworms. Its globular head is not so large as that of an average-sized pin, only measuring about a millimetre or $\frac{1}{25}$ inch, and it is armed by a double row of numerous hooklets, as well as by four large prominent sucking discs. The neck is fine and thread-like, and is succeeded by a long series of proglottides, which, about the middle of the worm, are broader than they are long; towards the extremity of the strobila these, when fully matured, are much longer than they are broad, measuring nearly half an inch in length and not more than half this in breadth. The number of proglottides or segments in a fully developed specimen may count up to nearly 1000. Upon examining a proglottis from the free end of the strobila the uterus may be easily identified, with its numerous branches—from seven to ten on each side—which subdivide. The ova are almost globular, number several thousands in each segment, and possess a thick outer shell with fine radiating striations or projections. In a perfectly mature embryo the six hooklets may be seen under the microscope.

A very important fact must always be remembered in the case of this worm. As already stated, the parasite requires two hosts, but occasionally man acts in the dual capacity, either by accidentally swallowing the ova of the *tænia* which inhabits his intestine after these have got under the finger-nails, or the ova may find their way into the stomach in water or in vegetables, as in the case of the pig. In either instance they lose their shell and develop into cysticerci, which are carried to distant organs. Having a special selective affinity for the eye and brain, they are not rarely found as minute bladders behind the retina, or in the vitreous, or in the anterior chamber of the eye, and sometimes on the membranes of the brain, or within its substance, or in the ventricles. Occasionally they are deposited as minute tumours under the skin.

TÆNIA MEDIOCANELLATA OR *T. SAGINATA*.—This parasite is known as the beef-tapeworm, the ox acting as the intermediate host. Owing to the absence of hooklets from the

head, which is only provided with four sucking discs, this worm is frequently termed the unarmed tapeworm, in contradistinction to the pork-parasite, which is often termed the armed tapeworm. The head is square, or more or less pyramidal, and about twice as broad at its upper wide part as that of the pork-tapeworm. Its total length seldom reaches beyond twenty feet; the writer possesses one specimen measuring twenty-five feet. The number of proglottides may reach 1,300. They are much larger than those of the pork-worm, the mature ones measuring three-quarters of an inch in length and only from a quarter to a third in breadth, whilst the segments about the middle of the strobila have about the same measurements reversed. The neck is long and filiform, and the uterus has much more numerous branches; the sexual opening projects considerably and is lateral, and the ova are larger and more oblong in shape than those of the pork-worm. The segments when passed out in the fæces are actively mobile, and may wriggle out of the anus independently of the act of defæcation and creep amongst the patient's body-linen or be found in the bed-clothes. Though the opportunities for auto-infection are thus frequent, this parasite never passes its bladder-stage in man. Its distribution is very wide, and in some districts, as South America, India, and Abyssinia, it is extremely common. In Great Britain this parasite is more frequently met with than even the *tænia solium*, and its presence seems to be upon the increase in Ireland, probably from the larger amount of meat and cattle imported of late years into the island.

BOTHRIOCEPHALUS LATUS.—This is known as the fish-tapeworm, the intermediate host being the pike or other fresh-water fish. Its distribution is confined to the lakes in Northern Italy, to the regions bordering upon the Baltic, to some parts of Switzerland, and Japan.

The strobila may measure as much as fifty feet in rare matured specimens; more frequently it is found of about half this length or under. A single segment is distinguished at a glance from the *tæniæ* already mentioned owing to its great breadth and shortness, measuring about the middle of the strobila half an inch across and about one-sixth of an inch in length, whilst at the tail or free end of the colony each segment is nearly square. The genital pores or openings are central, not lateral or marginal as in the *tæniadæ*; the uterus is unbranched, but is folded upon itself, and the total number of segments may count between 3,000 and 4,000. The head is flattened and grooved, oval or olivary in shape, and is provided with but two large suckers, having no hooklets. The ova are oval, and the shell is closed by an operculum or little lid situated at one end. The ovum is never passed from the body of the host in a developed or embryonic condition,

as in the *tæniadæ*, but in its egg-form, and requires to remain a considerable time in water or damp earth before it is hatched. When swallowed eventually by the fish, it is in the form of a ciliated embryo, which does not pass into the cystic stage, but retains its worm-like form in the muscles and viscera till swallowed by man.

Other species of *tænia*, as *T. nana*, which seems to be identical with *T. murina* of the rat, is found in the intestines of residents in Sicily and Southern Italy, and *T. flavo-punctata*, which is so named because of a yellow spot in each segment about the middle or upper part of the strobila, has been found in man; it is common in the rat, being identical with *T. leptcephala*. The intermediate host seems to be a species of beetle.

T. cucumerina or *T. canina* is rarely found in man, though common in the dog and cat. It is believed to pass its larval stage in the body of the dog-louse or of the flea, whence it is conjectured to reach the human stomach.

Tænia echinococcus is a small tapeworm which sometimes infests the intestine of the dog, but which is never found in man. Its cystic or bladder stage constitutes the formidable human disease known as hydatids, and described under its own title. The cysts differ from those produced in the cycle of the other *tæniadæ* by their compound nature, daughter or secondary cysts budding from the interior of the primary vesicle. It is worth notice that the simple cystic formation of *T. solium*, known as *cysticercus cellulosæ*, may under favourable conditions in man, as in the cerebral ventricles, attain the dimensions of a small plum.

Symptoms of Tapeworm.—Usually the parasites during their sojourn in the human intestine give rise to no disturbances of any kind, though, as in the case of the round-worms, all sorts of nervous phenomena have been attributed to them. The psychic disturbances, however, are often severe in neurotic subjects upon the discovery of the presence of the parasite after some segments of the worm have been observed in the *fæces*. As might be expected, these are more prominent in the case of *bothriocephalus latus*, which, owing to its great length, is sometimes passed in numerous segments coiled up together and forming a mass as large as a small egg. Probably owing to the large quantity of nourishment required by this parasite for its maintenance, the appetite of the host has been sometimes observed to be abnormally great, and a very considerable degree of *anæmia* has been also recorded in many cases. It seems that some toxic substances are occasionally produced by the parasite, which have been supposed to cause *urticaria* or erythematous eruptions; these may be the result of some temporary *dyscrasia* in the *tænia* itself from the absorption of drugs or ptomaines in the human

intestine ; and they may be the cause of the anæmia, since this is of the primary or pernicious-anæmia type.

Tapeworms may infest individuals of all ages, though they are comparatively rare in the very young.

The etiology of the condition is obvious : in all instances it is the result of eating pork, ham, bacon, fish, beef, or veal in a raw or imperfectly cooked state.

Diagnosis.—This is usually easy. An inspection of the segments passed in the fæces not only reveals the true nature of the case, but careful consideration of the points already emphasised serve to differentiate the species of cestode present. H. McKisack points out the importance of carefully scrutinising the objects declared by the laity to be tapeworms, since strips of mucus are often mistaken for the parasite. The writer has been several times consulted by persons who believed they had tapeworm, when his examination of the fæces revealed the fact that they had been eating raw seaweed or dulce.

The segments of *T. mediocanellata* not infrequently wriggle outside the anus, and have been discovered by a patient who never inspects his excreta.

Treatment.—The *prophylaxis* is obvious from the etiology of the condition. Thorough cooking of all food is essential ; rigid personal cleanliness in those infested by *tænia solium* is also necessary in order to prevent the development of the cysticercus. The close inspection of all pigs before curing should be insisted upon by municipal authorities : the cysticercus is usually easily detected in the animal by its visible presence under the tongue.

Of drugs the most reliable is male fern ; this seldom fails, when skilfully administered, in dislodging even *tænia solium*, which is by far the most difficult to expel owing to its double armature. The writer has elsewhere drawn attention to the variations in the strength of the liquid extract of this drug, owing to the variations in its environment, as in some climates it is almost inert. He has also emphasised what he believes to be a more constant cause of disappointment in its action, as *aspidium filix-mas* closely resembles *asplenium filix-femina*, from whose rhizome it can only be safely differentiated by its showing eight fibro-vascular bundles in the transverse section of its petiole base ; and the extract from this fern is liable to be unintentionally substituted for that of *filix-mas*.

Not less than 1 dr. and not more than 1½ dr. of the liquid extract of male fern in emulsion or in freshly made gelatin-capsules should be given after fasting or after the operation of a brisk purge in the early morning. This dose is usually sufficient to kill the worm and to cause its expulsion by brisk catharsis without the necessity of the further administration of purgatives. A careful search should be made for the head

of the parasite; if this be not expelled the segments will almost certainly be found again in the fæces before the expiration of about one hundred days.

Another precaution is necessary: if the worm be passed in a number of broken pieces, which is the rule, these should be carefully scrutinised. Owing to the gradual and regular alteration in the shape of the segments from the scolex to the free end of the parasite, it is usually easy, by placing the fragments in position, to determine in the *débris* whether one, two, or three worms had existed.

Other vermicides for the destruction of *tæniæ*—pomegranate root bark, pelletierine, kousso, turpentine, pumpkin seeds, areca nut, thymol, &c., are in use; but the male fern so seldom fails in the hands of the writer that he has little experience of their efficacy.

TETANUS

This, known also as Lock-jaw, is an acute toxæmia characterised by the presence of tonic muscular spasms in severe recurring exacerbations, caused by the introduction of the tetanus bacillus into the system (generally, if not always) by means of a wound.

Etiology and Pathology.—The disease is liable to occur at all ages and in all climates, though it is more prevalent in tropical than in temperate regions, and more frequent amongst the black-skinned races than the white. A specially named variety attacks infants through the infection of the stump of the umbilical cord by the bacillus, and sometimes, especially in the tropics, appears in epidemic form. Occasionally infection occurs at the uterine surface in the recently delivered, or in those recovering from abortion. Very occasionally a so-called '*idiopathic*' form of the disease is met with independently of obvious or visible abrasions or wounds, in which case the toxin is supposed to enter through some invisible breach of surface in the nasal, pharyngeal, or other mucous tract. In the great majority of cases the bacillus is introduced through a wound; and though deep and severe lacerated and punctured wounds are more liable to be followed by tetanus than simple ones, nevertheless the disease not infrequently follows the most trifling punctures, abrasions, or cuts, so that the primary site of inoculation is sometimes entirely overlooked or forgotten by the patient.

The disease has followed vaccination, probably by contamination of the site of the minute incisions after the introduction of the vaccine virus; but the lymph has been known to convey the disease in some rare instances. Tetanus has been many times conveyed in gelatin-injections, and the

bacillus has been found frequently in the gelatin used for this purpose. Tetanus has occasionally been produced by contaminated sera, hypodermic solutions, or infected needles; the disease has been traced to infected catgut used in operations. In some campaigns the mortality from tetanus amongst wounded soldiers has been very high, and gunpowder-wounds, as seen in the American annual July celebrations, are especially liable to be followed by the disease.

The *bacillus of tetanus* is the etiological factor in all cases. It is found commonly in garden-soil and in the sweepings from the streets of cities, to which it has been conveyed in clay or in the fæces of horses, oxen, or sheep. The bacillus has been found in earth at a depth of nearly seven feet from the surface. Its presence has been demonstrated in the dust of houses and in the crevices of the woodwork of floors and adhering to rusty nails, &c.

The microbe is a slender bacillus or rod measuring rather more in length than the tubercle bacillus (3 to 5 μ), but in suitable cultures it often grows in much longer filaments or delicate threads. It is motile till sporulation takes place, which occurs at one end of the bacillus, where a clear round point larger than the breadth of the rod develops, forming the so-called drum stick or pin-head bacillus. These spores are extremely tenacious of life and are unaffected except by high temperatures, withstanding water for an hour at the temperature of 175° F., and retaining their virulence when kept in the dry state from air and light for a long number of years. The bacillus is anaërobic, but not absolutely so, since its toxins have recently been proved virulent when the rods were cultivated under partially aërobic conditions; it flourishes best in ordinary media at about the body-temperature and in an atmosphere of pure hydrogen gas, and is destroyed with difficulty by solutions of the ordinary strengths of the antiseptics usually employed in surgery.

When the bacilli or spores have been introduced into the subcutaneous tissue they multiply, but remain *in situ*, and do not invade the blood-stream or visceral organs as other infective organisms do. They manufacture their toxin at the seat of inoculation; this is carried to the nerve-centres after its absorption, and produces the constitutional symptoms of the disease. The toxin itself has been isolated by filtration from the bacilli and spores; its virulence exceeds that of any other known poison, its toxicity being calculated to be between 300 and 400 times that of strychnine. A minute quantity of the sterile toxin injected subcutaneously produces all the symptoms seen in tetanus; but a noteworthy difference in its action from that of the poisonous alkaloids consists in the sterile toxin of tetanus possessing an incuba-

tion-period of considerable length. This period in the horse is as long as five days; and though the chemical nature of the pure toxin still awaits demonstration, it is believed on account of this incubation-period to be of the nature of an enzyme or diastase, which upon introduction into the living animal forms the really active toxin after acting upon some constituent of the body. It is also most interesting and significant that this incubation-period disappears when the blood or extracts made from the organs of animals dying from tetanus are directly injected into a healthy animal. The selective affinity between the tetanus toxin and certain parts of the nervous system is most remarkable. The toxin is found after death only in the central nervous system as a rule. Many observers are satisfied that during life the toxin travels only to the nerve-centres by the route of the peripheral nerves and not by the general blood-stream. However travelling, it finally reaches the motor cells in the anterior cornua of the cord and in the medulla, with the protoplasm of which in all probability it enters into direct chemical combination. The result of this chemical change flashes out in the increased action or hyper-excitability of this part of the central axis, and, according to Sherrington, of the sensory afferent limb of the reflex arc running in the posterior roots. It is a very striking fact that when brain-substance is mixed *in vitro* with the terribly virulent toxin of tetanus it produces no effect when injected into an animal. To explain this remarkable phenomenon Ehrlich invented his ingenious theory of 'side chains,' the toxin being supposed to contain two groups of molecules; one of these combines with a similar group in the nerve-cell, by means of which the toxin is fixed to the cell, the combination being slowly effected during the incubation-period after the injection. The second group of the toxin-molecules are supposed to induce hyper-excitability of the cell-protoplasm, and as the first set are cast off by the toxin in their regenerated condition they enter the blood, and, being still capable of combining with the toxin, they constitute the antitoxin. This theory is employed to explain the differences between natural and acquired immunity to the tetanus-toxin; in natural immunity the first-mentioned set of molecules in the nerve-cells are supposed to be absent: hence the corresponding haptophoric group of molecules in the toxin fail to combine with or to attach themselves to the nerve-protoplasm.

In order to test the value of this working hypothesis as applied to the phenomena of strychnine-poisoning, Professor Lorrain Smith and Dr. Dixon, at the request of the writer, performed a series of experiments with rabbits by hypodermically injecting strychnine which had been previously mixed with a fresh emulsion of spinal cord; but though a

marked degree of neutralisation could always be obtained, the animals usually succumbed to a double lethal dose of the alkaloid, no matter how large the dilution with spinal emulsion.

A rather remarkable fact has been reported by several observers, who found that when the spores of the tetanus bacillus are freed from any adhering toxin before being injected, the microbe fails to multiply itself within the animal-organism; moreover, the virus when administered by the mouth is innocuous. Metchnikoff observed that when the toxin of tetanus is mixed with the ovarian substance of the hen *in vitro* a chemical combination occurs like that which takes place when the virus is mixed with nerve-matter. When the toxin is injected subcutaneously in animals it is observed that localised spasmodic contractions are set up after a variable incubation-period before the general symptoms abruptly appear, but it has been demonstrated that the action of the poison is always centric.

Pathological Anatomy.—Any changes detected in the nerve-elements are almost certainly the result of the over-action of the cells; similar secondary histological changes are observable in strychnine-poisoning and allied conditions. The pathological or anatomical proofs of the disease may therefore be said to be entirely negative. The motor terminal plates and the muscles themselves are always normal. The writer once observed in a horse which recovered from a very severe attack of tetanus following a foot-wound, that a tumour, obviously consisting of a mass of permanently contracted scalenus muscle, remained for years, causing deformity in the neck, probably due to rupture of the fibres during a tetanic spasm; and in man, fracture of bones, dislocations of joints, and rupture of muscles have been occasionally recorded.

Symptoms.—These follow an incubation-period in man of between four and thirty days. In the majority of cases the first symptoms show themselves before the middle of the second week after the receipt of an injury, but the disease may manifest its presence as early as the first or second day after the wound. The symptoms consist of recurring spasms, which attack the voluntary muscles at irregular intervals. In some cases the spasmodic seizures are observed to commence in the muscles near the seat of injury, but in the majority of cases the first indication of the disease is afforded by a state of increased tension, causing stiffness of the neck-muscles, which is rapidly followed by rigidity or tightness of the jaw-muscles, preventing the opening of the mouth. This tonic contraction, popularly known as lock-jaw or trismus, is soon followed by a similar condition in other regions. The characteristic clinical manifestation of the disease is afforded

by the supervention upon this more or less permanently increased state of muscular tension of recurring attacks of intense spasms. The physiognomy of the disease is usually highly characteristic when a spasmodic attack is induced by asking the patient to protrude his tongue from between his stiffened jaws. The angles of the mouth are drawn out and the eyebrows elevated by the spasm of the facial muscles, constituting the hideous grin known as the *risus sardonicus*. The trunk-muscles in severe cases are involved almost simultaneously, the abdominal wall becoming as rigid as a board. The muscles of the back in a severe spasm contract so powerfully that the body may rest upon the occiput and heels (*opisthotonos*), or it may be twisted to one side (*pleurothotonos*), or in rare cases where the anterior trunk-muscles suffer most the body may be bowed forwards (*emprosthotonos*). These spasms are accompanied by more or less pain and a considerable degree of anxiety when of long duration, during which the patient is unable to breathe or cry out. They are excited by the least movement of the patient, as in swallowing, but they occur spontaneously when the body is in complete rest; their severity and duration are variable; as they pass off they leave the sufferer in an exhausted condition and bathed in perspiration. After the cessation of a spasm the muscles do not regain their normal condition, more or less rigidity always remaining, but sometimes in the fitful sleep which follows the exhaustion of a severe seizure they may appear relaxed. All the voluntary muscles in the body are usually affected, but the involuntary ones escape, the condition of the bowels, bladder, and heart being undisturbed. Spasm of the glottis may suddenly cause a fatal asphyxia, which, however, is more likely to be produced by a total fixation of all the respiratory muscles, rendering breathing impossible. The pulse is usually quick during a spasm and normal between the seizures, and the respirations are natural when the spasm passes off. The mind remains clear throughout, unless when in rare cases the temperature rises very high, in which case delirium may supervene. The temperature is often normal during the entire progress of the disease, though a slight transient rise may be usually observed during a spasm; but in some severe and early cases there is marked pyrexia from the beginning, and a fatal hyperpyrexia has been occasionally recorded.

In severe cases a fatal termination occurs about the evening of the third day after the onset of the symptoms, the patient succumbing to asphyxia during a spasmodic seizure, or he may sink from the intense exhaustion caused by the frequency and severity of the spasms. In milder cases the symptoms may last for many days, assuming a chronic type, which is very likely to end in recovery.

In the *idiopathic* cases—viz. those in which the disease has not been preceded by any discoverable wound or abrasion, the symptoms are more frequently of a milder type, and the probability of recovery is greater.

Tetanus neonatorum is certainly produced by the infection of the umbilical stump, and is not due, as formerly supposed, to the introduction of the virus by the respiratory tract. It is nearly always fatal within a few days, and, like the *puerperal* tetanus which follows parturition or abortion, it is commonly due to want of cleanliness upon the part of midwives. The disease in these forms is becoming very rare, owing to the almost universal practice of aseptic precautions, but in India both of the latter types are still met with, almost in epidemic form.

Cephalic tetanus is the name given to the disease when it follows an injury to the face or side of the head. In addition to the ordinary clinical features of the disease, there is paralysis of the facial nerve upon the injured side; the limbs often escape, the back muscles, the pharynx and the larynx bearing the brunt of the attack.

Diagnosis.—The presence of tonic contraction of the voluntary muscles, upon which severer spasmodic seizures superimpose themselves at varying intervals, when these are known to follow a wound received within a week or two, leave no doubt usually about the diagnosis. The chief diagnostic feature is the persistence of the milder contraction, rigidity, or increased tension in the muscles between the severer spasms. This is sufficient to eliminate *strychnine-poisoning*, in which, moreover, the jaw-muscles, if involved, only suffer at a late stage, though the entire body, including the hands, is convulsed. Between the spasms of strychnine-toxæmia there is complete relaxation, though the slightest touch or movement will bring on a convulsion. It is often stated that in doubtful cases an examination of the urine will settle the diagnosis by the discovery of strychnine; this is certainly not to be relied upon: the writer made a careful analysis of the urine in two cases of severe strychnine-toxæmia, and not a trace of the alkaloid was to be found in the urine in either case. The diagnosis cannot be settled by the duration of the symptoms, as some severe cases of tetanus may prove fatal in as short a period from the first symptoms as in strychnine-poisoning. The writer has witnessed severe opisthotonos in strychnine-poisoning as late as seven hours after the first tetanic convulsive seizure. These facts are of considerable importance in medico-legal cases, and the only conclusive evidence of tetanus being the cause of death in a rapidly fatal case is the discovery of the bacilli in the neighbourhood of the injury, or by inoculating into a small animal, as the house-mouse, a quantity of the secretion from the wound.

In all cases of tetanus the infection is a mixed one, but culture-experiments conducted by Kitasato's method are usually conclusive.

Hydrophobia is eliminated by the history and by the persistence of the muscular rigidity after the pharyngeal and other spasms have passed off. The mental state in hydrophobia is markedly different: the thought of drinking or even the suggestion of fluid is usually sufficient to bring on a severe attack of spasm.

Tetany is differentiated by the peculiarity of the seizures attacking the hands chiefly, and by the other characteristics mentioned in the next article.

Cerebro-spinal fever in its malignant type sometimes closely simulates tetanus in those acute fulminant cases which die in a few hours, and in which severe spasms affect the muscles of the neck, causing opisthotonos or orthotonos. The presence of an epidemic of the disease, of an eruption, of mental symptoms, with high temperature (though this is sometimes absent), but especially the presence of Kernig's sign and the demonstration of the diplococcus intracellularis in the fluid obtained by lumbar puncture, are conclusive. It must, however, be remembered that the meningococcus is often absent from the cerebro-spinal fluid during the first three or four days of the disease.

Hysterical attacks cannot usually be mistaken for tetanus owing to the preponderance of the mental or psychic disturbances, unless where the patient, having previously received a wound, fancies that symptoms of tetanus are supervening, in which case the other unmistakable stigmata of the neurosis are certain to be in evidence.

Prognosis.—This is always grave, and the gravity increases with the brevity of the incubation-period. Few cases recover when the symptoms of the disease set in as early as the fourth or sixth day. If the patient survive the symptoms of tetanus for four or five days the prospects of recovery brighten considerably, but death may occur from spasm of the glottis at any period. Behring maintains that the all-round mortality of the disease, which was 88 per cent. prior to the introduction of serum-therapy, has now been reduced to half this percentage. The prognosis should always be the very gravest in extensive lacerated wounds and compound fractures which have been obviously deeply contaminated with garden-soil or earth, though death is often known to follow apparently simple puncture-wounds.

Treatment.—*Prophylaxis* is of vital importance in the case of all wounds, especially in those contaminated by earth, and the thorough cleansing and disinfection of such should be rigidly attended to. Some surgeons insist upon the necessity of at once administering a full dose of the antitoxin

in all such cases, and there cannot be a doubt about the wisdom of such a procedure. Owing to the susceptibility of the horse to tetanus, veterinary surgeons now usually administer a full dose of the serum upon performing the operation of castration, and by this procedure the mortality of the operation has been markedly reduced.

Serum-therapy.—The brilliant discovery of Behring and Kitasato, that animals could be rendered immune to the tetanus bacilli by graduated small injections of the toxin, and that the serum from these protected animals also possessed immunising and curative powers, laid the foundation of the method of serum-therapy, which has already saved innumerable lives in diphtheria.

That this method of treatment often fails entirely in tetanus has only been too obviously demonstrated, but the unsatisfactory results in no way weaken the basis upon which serum-therapy rests. The clinical results correspond with the experimental; the efficacy of the method depends absolutely upon the time-limit. A dose of the antitoxin which is quite sufficient to save an animal if administered at the same time as a lethal dose of the toxin, has no effect whatever when injected after the lapse of even a brief interval. A much larger dose will, however, save the life of the animal when administered as soon as the first symptoms of the disease have shown themselves, provided the dose of the toxin has not been enormously greater than that necessary to destroy life.

In the case of the human subject, by the time that the first symptoms of trismus have shown themselves, the bacilli have been localised in the tissues around the original wound, where the toxins which they manufacture have been continually finding their way to the spinal nerve-centres, and probably have already become fixed there. The most that can, therefore, be expected from the injection of antitoxic serum is the neutralisation of any free toxin still existing in the circulating fluid and tissues of the body, and repeated injections may be expected to continue this antagonising action, so that further fixation of the toxin in the spinal cells is prevented. Tizzoni, Cattani, and Roux, however, maintain that the serum does not neutralise the toxin in a chemical sense, but that it acts by enabling the affected nerve-cells to continue their normal functions in spite of the presence of the toxin. All authorities agree that the only hope of success lies in the resort to the injection of the serum at the earliest possible moment after the first appearance of stiffness in the neck or jaw muscles. The chance of a satisfactory result diminishes with each hour of delay. Thus, in experimental investigations a dose of serum which would effectively protect an animal if given before inoculation with

the toxin, requires to be increased by about two thousand-fold if the operator waits till the first local spasms appear. If the delay extends to the period when the symptoms have become general, even this large amount must be again increased several hundred-fold. The serum has been injected by nearly every possible route, but the general consensus of opinion is in favour of the administration by the intrathecal method, through a lumbar puncture. The dose need never be less than 20 c.c., and the injections should be repeated as frequently as the symptoms warrant, the cerebro-spinal fluid being first withdrawn. Intracerebral injection possesses no advantage over the intraspinal, which is, moreover, much safer.

Bacelli's method of treating tetanus consists in hypodermic injections of a 3 to 5 per cent. solution of pure carbolic acid, 1 c.c. being injected every two hours or oftener. This plan is probably worthless by itself, but already there appears sufficient evidence to show that it may be employed advantageously along with the antitoxin treatment. Certainly it should be resorted to immediately in the neighbourhood of the wound, whilst the serum is being procured. Symmers's statistics show that the mortality in seventy-five cases treated by the carbolic-acid method only amounted to 23 per cent. The acid may be given by the mouth and rectum, as well as hypodermically, and may be pushed till slight symptoms of toxæmia appear. Blake recommends intrathecal injections of magnesium sulphate.

Other drugs formerly tried and abandoned are being again pressed into the service as adjuvants to the serum-method, as curare, morphine, chloroform, chloral, Indian hemp, &c., but the most promising aid to the antitoxin treatment would appear to be the adoption of Corning's suggestion of nearly a quarter of a century ago—*i.e.* of inducing spinal anæsthesia by cocaine. Many recent records have demonstrated that by intrathecal administration of eucaïne β the hyper-excitability of the cornual cells and of the sensory portion of the reflex arc contained in the posterior roots can be so profoundly influenced that the spasms are prevented. Morphine in small amount may be added to the eucaïne β before being injected through the lumbar puncture.

The condition of the wound demands immediate attention as soon as the spasmodic seizures occur, free disinfection and the prompt evacuation of all pent-up discharges being indicated. In all severe cases amputation of the part should be performed, when its peripheral localisation renders the operation practicable, as in the case of injuries of the fingers, hands, or feet. The general treatment of the patient as regards feeding, rest, and quiet should be conducted upon the same lines as in hydrophobia.

TETANY

This is an affection characterised by the presence of more or less continuous or paroxysmal bilateral tonic muscular spasms or cramps, affecting usually the peripheral parts of the extremities.

The disease occurs under very varying conditions and under many different forms, some of which are regarded as representing distinct affections.

Etiology.—Since the first complete descriptions of the disease, published in 1830 by Steinberg, and shortly afterwards by Dancc, a considerable literature has accumulated dealing with the etiology of the affection. It occurs chiefly in early life, the majority of the cases being met with before the end of the second decade. The different types of the affection are markedly differentiated by age; thus, the form usually associated with rickets is met with in early childhood, whilst that occurring in artisans of sedentary occupation is oftenest met with about middle life. The influence of sex is seen when the disease occurs in early childhood, most of the cases being males; after twenty, nursing or pregnant women supply the majority of examples of the affection, as in the form described by Trousseau as 'Nurse's Contracture.' The disease is most common in the cold spring or winter months, and this seems especially true of the cases occurring in otherwise healthy artisans. It is liable at these seasons to appear in *epidemic* form in sedentary workmen, in the inmates of prisons, asylums, and schools. The geographical distribution of the affection is interesting: in certain cities, and notably in Vienna, the disease is more frequently met with than in Berlin or London.

Gastric diseases are sometimes associated with tetany, and the condition described first by Kussmaul as gastric tetany is seen in a typical form in some cases of dilatation of the stomach; a similar variety is sometimes observed in intestinal disorders, chronic diarrhoea, dyspepsia, &c., and in children or adults who are the hosts of any of the usual intestinal parasites.

Tetany has been several times observed to arise during convalescence from cholera, the various exanthemata, and influenza. Poisons, as ergot, lead, chloroform, alcohol, morphine, and urea, have been credited with the production of symptoms of tetany.

Thyroid-gland disease sometimes, but more frequently total extirpation of the gland or of the parathyroids, has been followed by tetany. The condition has been also found associated with such pathological states as exophthalmic goitre, syringomyelia, and cerebral tumours.

The above etiological factors have been taken as a basis for the classification of tetany into seven distinct groups by Frankl-Hochwart, but till the pathogenesis of the condition has been worked out it appears safer to regard tetany as a distinct entity or symptom-complex, in which any one or more of several etiological factors may act as exciting or predisposing causes.

Pathological Anatomy and Pathogeny. — The slight changes sometimes observed in the cord and brain, as œdematous or hyperæmic areas, slight sclerosis or myelitis in the cervical region, and less frequently in the lumbar enlargement, cannot be regarded in any sense as characteristic of the condition, since they are much more frequently entirely absent. Various theories have been invented to explain the pathogeny of tetany, but most of these have been abandoned for the *toxæmic* hypothesis. This is the generally accepted view, the condition being now regarded as an intoxication, though differences of opinion exist as to the site of manufacture of the poison, some authorities adhering to the view that the toxin producing the symptoms known as tetany is formed in the stomach or intestines. Bouveret and Devic have recently isolated from the gastric secretion of a tetany-patient a substance which they regarded as a peptotoxin, which produced the characteristic spasms when injected into a dog. Though these observers considered that this toxic product was the result of the formation of an excessive amount of free hydrochloric acid, it cannot be denied that the symptoms of gastric tetany occur sometimes in dilatation of the stomach where, though there is great excess of butyric and other organic acids, the free hydrochloric is greatly diminished or absent. Much attention has been directed to the study of the tetany which follows extirpation of the thyroid gland in animals and man, and the important fact has been demonstrated that the spasms characteristic of the disease only follow when the parathyroids have been removed; hence the theory that the parathyroids have the power, under normal conditions, of destroying some toxic substance whose retention poisons the nervous system. Should such substance, however, be produced in large quantity in a dilated stomach, the parathyroids might prove unable to cope with it, even when these are normal and active. It is a significant fact, however, in this connection that thyroid tetany is more effectually relieved by the administration of thyroid than of parathyroid-gland substance.

An interesting study is that which might be considered as the '*pharmacology*' of the poison: how does it act, or upon what portions of the nervous system is its influence exerted? The site of its action can only be conjectured, owing to the anomalous nature of the symptoms and the negative char-

acters of its pathological anatomy; but in all probability the poison selects chiefly the anterior ganglion-cells of the cord, though its affinity for these is much less marked than in the case of the tetanus-toxin and of strychnine. It probably also acts upon the peripheral nerves to a lesser extent.

Drummond sums up the pathogeny of tetany as that 'of a group of symptoms produced by the action on the nervous system of a toxic substance or substances, which arise under different conditions, and whose nature is unknown.'

Symptoms.—The striking clinical feature is the presence of tonic muscular spasms which most commonly attack both hands, and less frequently both feet. The spasm may be immediately excited by some severe muscular exertion, or may occur in the temporary muscular exhaustion following a prolonged effort. In some cases a marked mental emotion may induce a spasm, and whether this is the explanation of the spasms in gastric tetany being often induced by an attempt to pass a tube into a dilated stomach, is not easily determined; probably the hyper-excitability of the motor apparatus is intensified by psychic causes.

In the hands the spasm or cramp generally leads to the action of the muscles bringing the fingers, palm, and wrist into a highly characteristic position or attitude. The fingers are flexed at the metacarpo-phalangeal joints, and extended at the second and terminal joints; they are pressed closely together, their tips forming the apex of a cone, whilst the palm is deepened by the approximation of the thenar and hypothenar eminences, so that the hand assumes the position known as the obstetric or accoucheur's hand, only that the thumb is not only adducted but powerfully flexed upon the palm. The wrist-joint is flexed and usually deviated strongly to the ulnar side, and should the spasm extend upwards the fore-arms are pronated, the elbows flexed, and the upper arm held closely to the sides of the chest. An almost similar position of the entire upper extremity has been witnessed by the writer several times when a person holding the poles of a strong electrical battery had received a powerful induced current which prevented him dropping the terminals. Occasionally the hand is contracted into the 'fist' form, with all the joints flexed strongly upon the palm. The 'griffin-claw' shape of hand is very seldom assumed in the attack. In the pedal spasms the toes are flexed and adducted, the foot assuming the position of talipes, with the sole inverted and the ankle-joint extended; and when the cramp extends upwards the thighs may be strongly adducted and the hips flexed, or the knees either flexed or extended.

In severe cases the spasms may be widespread, the facial muscles exhibiting the risus sardonicus seen in tetanus, and even trismus may occur, though this is rare according to all

observers ; strabismus sometimes occurs, and the tongue and laryngeal muscles are occasionally affected. When the neck-muscles are thrown into spasm, opisthotonos or marked retraction occurs ; but more frequently in such severe cases the head is flexed upon the chest, and the spasm may involve the thorax, diaphragm, and abdominal muscles, causing serious difficulty of breathing, and lividity.

The duration of a spasm and its severity vary greatly ; in mild cases the seizure only lasts for a few minutes, the paroxysms returning again in a few hours. In severe cases the duration of the spasm may be a period of many hours or a few days, and even after the disappearance of the cramp, if the muscles be carefully examined, a minor continuous tonic contraction can be made out, which sometimes lasts till the advent of the next paroxysm. Hence the types of the spasm have been sometimes described as continuous, remittent, and intermittent, but these forms may be all marked in the same case at different times. As pointed out by Risien Russell, the spasm is never so severe in the continuous type, which is most frequently met with in children.

During the paroxysm the cramped muscles are found to be hard and firmly contracted, so that a considerable degree of force is necessary to restore the flexion or extension of the joints, which speedily snap back again into their abnormal position, sometimes with an invasion of the cramp in proximal muscles not previously contracted. Should the patient fall asleep, the spasm, though lessened in intensity, often does not pass away, and in some cases the paroxysms may even commence during sleep.

Trousseau's sign is of considerable diagnostic value, and when present is almost pathognomonic ; it consists in applying pressure over the main vessels of the limb during the interval between the paroxysms, when an attack of the typical cramp in the muscles formerly involved will be reproduced. It is almost certainly due to the irritation of the nerves, which usually run along with the great vessels, and not to any obstruction of the circulation, as formerly supposed.

Chvostek's sign is also of great diagnostic significance, and is based upon the constantly present hyper-excitability of the muscles and of their motor nerve-supply. It is elicited by making a smart tap on the nerve supplying a muscle, or upon the muscle itself, during the interval between the paroxysms, when a marked contraction of the latter will follow. It is most obvious when the tap is made over the facial trunk or one of its chief branches, in which case the facial muscles are thrown into firm cramp, even when these were not previously involved in the paroxysms.

Erb's Sign.—The electrical reactions are curiously altered, the chief characteristic change being, in addition to the

generally increased excitability of the nerves to the galvanic current, a more marked response with the anode, when the current is closed or opened, the contraction being a prolonged tetanic one. As Russell points out, this anodal-opening tetanus exists in no other condition but tetany, though it is not invariably present. The altered electrical reaction is best marked in the ulnar, and seldom if ever observed in the facial. The increased excitability to the galvanic current is almost invariable, but to the induced current the hyper-excitability is much less frequently marked.

There is nothing distinctive as regards the condition of the reflexes; the knee-jerks, when these can be tested (there is often difficulty in doing so owing to the presence of the muscular spasm), may be either increased, diminished, or absent.

Akin to the above phenomena depending upon the condition of hyper-excitability in nerves and muscles, is the induction of a paroxysm when the stomach-tube is introduced for the purpose of washing out the organ in cases of dilatation.

There are usually but slight constitutional symptoms, only the breathing and pulse being quickened during the paroxysms. In severe attacks dyspnoea, cyanosis, and laryngismus may, however, be formidable when the respiratory and laryngeal mechanism is interfered with. In the tetany occurring amongst healthy artisans, and sometimes described as 'workman's,' 'shoe-maker's,' or 'tailor's' cramp or tetany, there is usually a mild fever of two or even three weeks' duration, and a similar febricula has been observed in the epidemic form occurring in asylums and schools.

The urine shows often a trace of albumin, especially when the disease occurs in its severe form in adults; mucin, indican, sugar, peptotoxin-substances, and increase of phosphates have been often observed.

Vaso-motor phenomena are frequently met with, the most constant sign being a slight solid oedema and redness of the dorsum of the hands and feet, which disappears during the paroxysms, and remains in the intervals. Rarely do trophic signs appear, and when present only amount to slight swelling of the joints or tendinous sheaths, and, like the spasms and oedema, are always bilateral.

Sensory disturbances are as a rule associated with the paroxysmal attacks, and prodromal numbness or formication may be experienced a short time before the advent of cramp. Pain is often very severe; when the muscular contraction is prolonged and intense it may shoot up into the proximal muscles and joints, and it may remain in a mitigated form for a variable period after the departure of the spasm.

The progress of the disease is most variable, and the length of the period during which the paroxysms continue to recur

varies greatly, depending apparently in many instances upon the nature of the associated condition or disease. In children the duration of the disease is usually short, especially when the type of the spasms is continuous. In the tetany of pregnancy the attacks continue to recur till parturition takes place. In thyroid tetany and in the gastric form the attacks usually continue to recur till a fatal issue results. After the disappearance of all paroxysms they are liable in some cases to be brought on by exposure or prolonged muscular exhaustion, or to return during a subsequent pregnancy or lactation period.

Diagnosis.—In a marked case error in diagnosis can hardly occur if the characteristic recurring attacks of bilateral tonic spasm, affecting the peripheral portions of the extremities, are recognised. The signs of Trousseau, Chvostek, and Erb are pathognomonic if present; sometimes a sign associated with the name of Hofmann and not previously mentioned may also be demonstrated: this consists in the production of paræsthesia when a sensory nerve is pressed upon, owing to the hyper-excitability of the sensory fibres.

Rickets affords a difficulty in diagnosis only of a theoretical kind; most authorities agree that the carpo-pedal spasms met with in the second year of life are to be recognised as tetany occurring in association with rickets. Even when no other rachitic manifestation can be detected, the presence of the underlying error in assimilation may be safely accepted, if every recognised cause of tetany be absent, as convalescence after an eruptive fever, gastric or intestinal lesion, &c.; Escherich regards the sole presence of laryngismus stridulus as conclusive of tetany. In this connection the important fact should be kept in mind that tetany is most frequently met with in the mild and not in the severe type of rickets.

Tetanus is differentiated with ease, unless in those severe cases of tetany where there is spasm of the neck-muscles and opisthotonos present with trismus. The absence from the history of the case of an injury, the distribution of the early spasms, these being confined to the hands and feet, and the time at which trismus first appears in tetany are characteristic, since trismus is always the first sign in tetanus, and if present at all in tetany is invariably a late manifestation. The feet and hands almost invariably remain unaffected in tetanus, and the muscular spasm is continuous, with severe exacerbations. Risien Russell points out that in tetanus the extensors, supinators, abductors, and external rotators are in action; whilst in tetany the muscular groups involved are the flexors, pronators, adductors, and internal rotators. Moreover, the tendency towards a fatal result in the one disease contrasts with the usually safe course of the other, unless laryngismus supervenes. In *tetanus neonatorum* the attack sets in during

the first week of life with the symptoms of trismus and the other manifestations of true tetanus, tetany never being met with at this early period of existence.

Tubercular meningitis usually sets in at a later stage in childhood than rachitic tetany. There is an insidious onset, during which the symptoms slowly deepen in gravity as the cerebral disturbances manifest themselves in severe headache, vomiting, ocular paralysis, optic neuritis, irregular respiration and pulse, delirium, and coma; spasm, when present, never betrays itself in the characteristic attitude of the hand seen in the recurring spasms of tetany.

Cerebro-spinal Meningitis.—Fever is generally present; the tonic spasm is usually, at first or throughout, limited to the neck and back muscles; there is severe pain radiating along the course of the nerves and felt independently of muscular contraction; a rash is often present, cerebral symptoms supervene, Kernig's sign is present, and the specific diplococcus intracellularis is found in the fluid from a lumbar puncture.

Hysteria is eliminated in the diagnosis of the tetany of adults by the bilateral or symmetrical distribution of the spasms, by the absence of the usual hysterical stigmata of anæsthesia, hyperæsthesia, paralysis, &c., but in cases more closely simulating or occurring in association with hysteria the most important diagnostic point is the demonstration of the signs of Trousseau, Chvostek, or Erb, which prove that the condition of tetany is present.

Epilepsy of the Jacksonian type is easily differentiated from tetany, since the spasm is unilateral always at the beginning, and as soon as it spreads so as to become bilateral or general, consciousness is lost and more or less weakness remains for a time in the groups of muscles the seat of the spasms, which latter, moreover, are both tonic and clonic in character. Other evidence will often be found of the lesion causing the localised convulsions, as severe cephalalgia, optic neuritis, &c. From ordinary *epilepsy* the diagnosis is still easier, since in the general or bilateral tonic and clonic convulsions consciousness is always lost and, as in the Jacksonian type, the spasms are of very brief duration.

Prognosis.—In the tetany of childhood death is a rare event, and only occurs when laryngismus or other complication affects the respiration. In the tetany following or supervening upon the exanthemata the outlook is less bright, though as a rule good. Even in pregnancy or lactation tetany the spasmodic seizures as a rule disappear with the underlying cause. The prognosis of tetany in the adult from whatever cause is not so favourable as in childhood, though the tetany occurring in sedentary workmen seldom proves fatal. The most serious prognosis must be

given in the form which occurs after the removal of the thyroid, though thyroid-gland feeding usually postpones or averts the fatal issue as long as the treatment is continued. Gastric tetany is always fatal unless the dilatation be promptly treated by gastro-enterostomy; but the symptoms often end in death before any operation can be performed. In these latter cases a serious question arises about the passage of a tube to remove the toxic stomach-contents; probably the best line of action in such an emergency is to wash out the stomach under chloroform-anæsthesia to diminish the risk of immediately aggravating the spasmodic paroxysm.

Treatment.—In all cases the underlying or associated condition should be removed as speedily as possible. Thus rickets and lactation have their own clear indications for treatment; and the tetany of gastric origin should be promptly met by lavage, using a mild solution of creosote or potassium permanganate to wash out the greatly dilated stomach in which the peptotoxins are being manufactured. The risk of using the stomach-tube when the condition of tetany is already marked by an active paroxysm has been just referred to. As a substitute for lavage the patient may be made to drink large draughts of tepid water in order to induce vomiting. The abdomen should be opened as soon as the patient's physical state warrants, and a communication between the intestine and posterior part of the stomach effected. The lavage, if relied upon alone, is almost certain to end in the patient passing away in some not far-off attack. Mayo Robson states that the final appropriate and only rational treatment of gastric tetany should be surgical, and W. A. Mackay and Ian Macdonald have recently successfully treated a severe case of gastric tetany by gastro-enterostomy. This is the twelfth case reported, with but three deaths from post-operative complications.

Thyroid tetany should be treated by thyroid-gland feeding, which Purves Stewart points out is more efficacious than the administration of parathyroid tissue.

The tetany of the puerperal period usually also yields to thyroid feeding, though this agent possesses little or no influence over the tetany associated with other conditions.

For the relief of the spasm a tepid or warm bath is often efficacious, and in rachitic tetany a dash of cold water when the patient is in the bath often acts promptly. A hot sponge over the larynx may be tried when cold affusions or applications of ice to the spine fail. Chloroform-inhalation or nitrite of amyl often relieves formidable spasm threatening asphyxia, and massage may be performed with striking benefit when the patient is under an anæsthetic. Pilocarpine hypodermically often speedily relieves the spasm, probably by rapidly causing the elimination of the poison; and with the same

intention other diaphoretics and diuretics are recommended. Flushing out of the colon with copious enemata is also useful, and emetics and brisk purgatives are advocated.

In the chronic cases the best routine, in addition to the above remedies, will be found in large doses of bromides combined with chloral hydrate.

THOMSEN'S DISEASE.

(See under MYOTONIA CONGENITA, on p. 1073.)

THREAD-WORMS. (See under OXYURIS, p. 1153.)

THROMBOSIS

This is the name applied to the condition in which a blood-clot forms in the arteries, veins, or cavities of the heart during life. The red thrombi consist of the ordinary ingredients found in normal blood-clot—viz. red and white cells, with fibrin; but in the composition and formation of the so-called white thrombi blood-platelets have been demonstrated to play a prominent rôle; in both cases, however, the essential process is a coagulation of the blood.

Etiology.—There are three recognised factors in thrombosis, any one of which may be able to establish the condition; all three may be acting in conjunction in some cases. These are (1) an altered condition of the lining membrane of the vessels or heart whereby its smooth polished surface is destroyed, the result tending towards deposition of blood-clot upon the altered endothelium; this is the usual explanation of the thrombosis seen in atheromatous vessels. Inflammatory changes of an acute nature act in the same way upon the vascular lining, and this cause probably furnishes the majority of the cases of venous thrombosis so commonly found as a part of the process of phlebitis. Doubtless many of these may be of infective origin, and such is probably the explanation of the vegetations forming on the surface of the cardiac valves by the growth of the diplococci which cause acute rheumatism. According to some authorities, microbes play a chief part in the thromboses called 'marantic' by Virchow, which are liable to form in anæmic states, in grippe, typhoid, and other fevers. In these latter cases there is often a secondary or mixed infection of streptococci found in the thrombi; these micro-organisms are the primary factor in phlegmasia alba dolens.

That bland thrombi may form independently of infective agents is proven by the clotting which occurs in veins which have been injured by blows. Thrombosis is common in both arteries and veins in the immediate neighbourhood of septic or necrotic areae.

(2) Stagnation of the blood-stream is a factor in some cases ; it is the main element in the rapid growth or increase of the thrombus when this latter has once been started by any cause. Eddies or side-currents greatly intensify the clotting.

(3) Changes in the composition of the blood are believed by many to play an important part in the tendency to thrombosis, the old theory of the excessive formation of fibrin giving way to the new one of increase in the number of blood-platelets. This latter is the supposed cause of the thrombosis so often seen in chlorosis. It is, however, possible that alterations in the blood may act by first involving the vascular walls. The thrombus, once started, tends to increase by the deposition of fibrin and corpuscles till complete blocking occurs ; or it may become detached, when the loose part is known as an embolus, and may be swept to a great distance by the blood-stream.

The loose or free coagulation-products found in the heart, chiefly in the left auricle in mitral stenosis, are known as ball-thrombi. They are generally associated with the presence of adherent thrombi in the auricular appendix, not necessarily produced by micro-organisms.

The symptoms of thrombosis are such as are caused by the shutting off of the blood-supply in the arteries, as described under 'Embolism.' In the veins the symptoms may be latent, but local dropsy is likely to occur ; and in thrombosis of the femoral vein much pain, numbness, passive congestion, and anasarca may supervene, though gangrene hardly ever follows. In mesenteric thrombosis necrosis is common, and the condition closely resembles that found in blocking of the superior mesenteric artery (p. 360).

Venous thrombosis is liable to lead to pulmonary embolism and sudden death, and cardiac thrombosis in the right heart produces the same result. When smaller branches of the pulmonary artery are blocked infarcts in the lung are produced. Thrombosis in the left heart, in the pulmonary veins, and in the arteries leads to symptoms and results already described under 'Embolism' (p. 356). Thrombosis of the cerebral arteries and venous sinuses is described under 'Cerebral Thrombosis' (pp. 167 and 171).

There are two well-recognised forms of thrombosis of the *portal vein*. Where the blocking is caused by a thrombus which is not infective the resulting condition is described as simple or *adhesive phlephlebitis*. It occurs as the result of

coagulation of the blood in the portal vein from disease of its coats or from the stagnation of the blood-stream in cirrhosis, perihepatitis, syphilis, tumours, chronic peritonitis, &c., or from the extension of thrombosis from the intestinal, splenic, or coronary vein of the stomach.

The symptoms may be entirely latent in some cases, but marked examples of the condition have been observed commencing with a sudden onset of portal obstruction, as evidenced by hæmatemesis, tarry stools, splenic enlargement, and ascites, and generally with dilatation of the superficial abdominal veins. The hæmorrhage may be rapidly fatal; if not, the ascites will probably speedily become extreme and re-accumulate very soon after tapping. In rare cases the anastomoses formed by the superficial abdominal veins may establish the collateral circulation.

When portal thrombosis is the result of septic causes introduced from the region discharging blood into the branches of the portal vein the resulting condition is a form of pyæmia known as *suppurative pylephlebitis*. It may occur in every form of ulceration affecting the stomach, or bowel in any portion of its entire length; thus the origin of the infection may be an anal ulcer or an appendicitis, dysentery, typhoid fever, or pelvic disease, ulceration of the biliary passages caused by gall-stones, splenic or pancreatic disease; tuberculosis of the liver or hydatids may supply the infective material. (See under 'Liver Abscess,' p. 818.)

In all cases the results are the same: thrombi form in the branches of the portal vein; these rapidly break down and become purulent, and are carried by the blood-stream to all parts of the liver, setting up multiple or pyæmic abscesses. The symptoms are those of pyæmia, in addition to which pain and tenderness over the liver and some enlargement of the organ, with slight jaundice, are present; when the obstruction involves a large number of branches there is swelling of the spleen and, if the patient survive long enough, some ascites.

The clinical picture of pyæmia *plus* hepatic symptoms in a case without external wound is the feature in diagnosis.

The prognosis and treatment are those of pyæmia.

As a rule, the symptoms of thrombosis are slower in their onset than is the case in embolism, and this is often very marked in cerebral cases; but a 'riding' embolus, especially when trapped at the bifurcation or branching of an artery, may become the nucleus of an extensive secondary thrombosis, which gradually and slowly blocks many neighbouring branches.

The nature of the thrombus will seriously influence the result: thus septic growths tend to soften, break down, and suppurate; whilst bland thrombi may also soften and become

absorbed, or be replaced by an organised connective-tissue formation, which may completely and permanently obliterate the lumen of the artery or vein. Sometimes free phleboliths or arterioliths are found in vessels the seat of old thrombi; they are caused by calcification of white thrombi.

THYMUS GLAND, DISEASES OF

The mystery which still surrounds the function or functions of the thymus is not being lessened by the patiently collected series of diseased conditions in which marked changes of a hypertrophic or atrophic state of the gland are being found. Thus the thymus has been found persistent or hypertrophied in such different diseased states as acromegaly, exophthalmic goitre, lymphatism, thymic asthma, myasthenia gravis or asthenic bulbar paralysis, and occasionally also in leukæmia. Atrophy of the gland has been found in marasmic conditions and in rickets; though in the condition known as lymphatism, which has been supposed to be due to hypertrophy of the thymus, rickets is also very commonly or generally present. Basch corroborates the important results obtained by Friedleben and Mettenheimer, who produced rickets in animals by totally extirpating the thymus; and other observers have reported success in the alleviation of human rickets by administering the dried gland-substance from animals. No observations, as far as the writer knows, have been yet made upon the few successful cases in which the gland has been extirpated for the relief of thymic asthma in infants by Ehrhardt. Under 'Lymphatism' the condition known as Thymic Asthma will be found described upon page 944, along with Thymus Tod.

THYROID GLAND, DISEASES OF

The only important diseases of the thyroid gland of purely medical interest are Myxœdema (p. 1088) and its allied condition of Cretinism (p. 251), and also Exophthalmic Goitre (p. 496), which are described under their own headings. Goitre or Bronchocele is the term applied to all enlargements of the thyroid gland of hypertrophic nature—i.e. those which are not inflammatory or malignant—and these are mainly of surgical interest. In the most typical variety, known as *simple parenchymatous goitre*, there is a true uniform hypertrophy of the entire gland, which commonly is met with in *sporadic* form. A *vascular* form is also recognised, in which the enlargement is confined to a dilatation of the blood-vessels without true hypertrophic glandular growth,

An *adenomatous* form is also met with, which is best regarded as a tumour of the thyroid.

Endemic goitre may commence as a simple parenchymatous hypertrophy of the entire gland, but the tendency later on is usually towards cystic degeneration, with proliferation of the fibroid elements, so that enlargements of varying dimensions ultimately form, which are known as *cystic* and *fibro-cystic goitres* in proportion to the amount of cyst-formation or fibrosis present; these growths, according to Page, are always encapsuled. This form of goitre is most commonly met with in women, and commences usually after puberty; the cystic development may occur also in simple adenomata of the thyroid, in which cases the cyst is usually single. The endemic character of simple goitre is recognised by its prevalence in certain localities, especially in the valleys between mountain-ranges, as in some parts of Switzerland, the Tyrol, and Northern Italy, where it may exist in association with cretinism, the children of goitrous parents being frequently cretins. In England the localisation of the condition is seen in the popular name of 'Derbyshire Neck.' It also occurs in flat regions in India, Italy, and Central Asia, and is still common about the shores of some of the great lakes in North America and Canada.

The etiology of endemic goitre is intimately associated with the nature of the water-supply, but the old theory of its being caused by the presence of lime-salts in the drinking-water has gradually given way to the microbic theory of the water-contamination since the discovery that boiling renders the water safe. There seems little to be said for St. Lager's hypothesis that the disease is the result of sulphides of iron and copper in the water.

Symptoms.—These are only such as are due to the mechanical pressure exerted by the growth upon the neighbouring structures, but especially upon the trachea, and only very rarely upon the recurrent laryngeal nerves or the veins. Dysphagia from pressure upon the œsophagus is only experienced in large tumours, dyspnœa being the chief clinical symptom in the majority of cases where the tumour is sufficiently large to cause any disturbance; in all cases the patient complains of a feeling of fulness in the neck. The shape and size of the tumour vary usually with the degree of cystic transformation. In adenomatous cysts the lop-sided nature of the enlargement is apparent, the fluid being on either side of the middle line or in the isthmus. A simple adenoma without cystic change has the same localised distribution, but it is smooth, rounded, and elastic, without fluctuation. In the simple parenchymatous form of the disease the tumour presents all the normal characters of the thyroid, save in the great dimensions caused by the uniform hypertrophy

of all parts of the gland—its two lobes and isthmus. A marked characteristic of all goîtres is that they move with the larynx in the act of swallowing. In large cystic goîtres there is occasionally seen an extensive hæmorrhage into the growth, which suddenly increases its tension and may lead to the rapid production of grave dyspnœa, and even asphyxia. In former years, before the dangers of tapping these cysts were recognised, the writer several times witnessed uncontrollable hæmorrhages follow the use of the trocar.

The temporary enlargement of the thyroid, which often occurs in women at the menstrual period, in chlorotic girls, and in the earlier months of pregnancy, is not regarded as falling under the designation of goître or bronchocœle. In these cases the increase in size is probably due to the dilatation of the blood-vessels of the gland.

Treatment.—In endemic goître the tumour may be expected to remain as long as the patient continues to use the drinking-water and to reside in the goitrous district. Boiling of the water or removal of the patient from the district may be confidently expected to cause its disappearance in the early stages of its growth. In simple parenchymatous goître excellent results are often obtainable by the administration of thyroid extract in moderate doses; but some cases fail to respond to this treatment. Iodine internally and locally, both by deep parenchymatous injection and applied to the skin, occasionally proves efficacious, but the injections are not free from serious danger. When the tumour causes great deformity, dyspnœa, or pain, surgical measures are indicated; these vary with the nature of the case, and numerous plans are employed, which may be found described in the author's 'Dictionary of Treatment' (4th ed.), pp. 324 to 329.

Malignant disease of the thyroid gland occurs either as the alveolar epithelioid variety or as a sarcoma. It may supervene upon an old goître or start in a healthy gland. The hard, irregular, painful, and fixed nature of the growth is usually easily recognised, and in rapidly growing cancer it soon tends to infiltrate neighbouring structures and to involve the recurrent laryngeal nerves, causing vocal-cord paralysis. In simple goître the difficulty of breathing is nearly always due to direct pressure on the trachea; in cancer it is of laryngeal origin usually. Sarcomatous growths are softer, the rate of enlargement is slower, and there is often a marked tendency towards cystic formation.

The treatment is entirely surgical, and consists in the early removal of the growth or of the entire gland; but once the disease has extended beyond the capsule of the gland extirpation is useless.

Inflammation of the thyroid gland, or *acute thyroiditis*, is a rare complication of typhoid fever, scarlatina, parotitis,

acute rheumatism, influenza, or scarlatina, and it is occasionally the result of surgical procedures, as iodine-injections, &c. Its advent is usually sudden, with a distinct rigor and high fever. One lobe, or the entire gland, becomes very tender, painful, and enlarged, so that neighbouring structures are pressed upon so suddenly that they have not time to accommodate themselves to the rapid pressure; dyspnœa, dysphagia, and vocal-cord troubles may arise from a degree of enlargement which in ordinary goîtres might produce no inconvenience. The skin may be early involved over the inflamed portions of the gland; resolution usually follows within a few days, but occasionally the affected lobe or the entire gland may suppurate or slough, and myxœdema or exophthalmic goître has been known in rare instances to follow.

In all operations for the relief of thyroid-gland disease the surgeon now realises the importance of leaving a portion behind when possible, total extirpation being liable to be followed by myxœdema.

Much diversity of opinion still exists regarding the functions of the *parathyroids* or accessory thyroid glands, which in animals like the cat and dog can be removed independently of the thyroids; but in monkeys they are inseparably incorporated with the latter, and are always extirpated when the thyroids are removed. In man accessory thyroids have been found in the vicinity of the thyroid, about the root of the tongue, and even in the vicinity of the aortic arch, and it is believed that when myxœdema does not follow after the removal of the thyroid the result is due to the presence of these accessory glands, which then take on the functions of the extirpated organ. The small mass of lymphoid tissue constituting the accessory thyroid at the base of the tongue, and known as the lingual thyroid, has been found the seat of changes resembling those observed in parenchymatous goîtres.

The supposed function of the parathyroids has been referred to in the discussion of the pathogeny of tetany. Some observers believe that they neutralise the action of the toxic substances which are produced in the metabolic changes due to the influence of the thyroid internal secretion, though Blum maintains that the thyroid functions as an excretory organ, eliminating iodine from the central nervous system.

TICK FEVER

This condition is referred to under 'Piroplasmosis,' p. 1358.

TINEA. (See under RINGWORM, p. 1531.)

TINNITUS.

(See under AUDITORY-NERVE DISEASE, p. 97 ;
and under VERTIGO, p. 1819.)

TONGUE, DISEASES OF

These are mainly of surgical interest ; acute superficial glossitis is nearly always a part of the general condition described under 'Stomatitis,' and the so-called 'geographical tongue' may be regarded as a chronic form of superficial glossitis, and allied to eczema.

Leucoplakia or *Leucokeratosis* is described under its own title, and its relationships to syphilis and cancer are referred to upon page 813 and upon page 1658.

Paralysis of the tongue is described under 'Paralysis, Peripheral,' p. 1210.

Syphilis of the organ will be found referred to on page 1658.

TONSILS, DISEASES OF

Those of purely medical interest are already embraced in the description of 'Pharyngitis' on page 1287.

ACUTE TONSILLITIS

It is rare for the acute inflammatory action to be confined to the tonsils in any case of acute tonsillitis, neighbouring structures, as the mucous membrane of the pharynx and palate, and also the uvula, being almost invariably invaded. Hence the confusion between the classification of sore-throats, pharyngitis, and tonsillitis. An acute pharyngitis undoubtedly is seen in which only the mucous membrane over the tonsils is apparently engaged ; this is usually of the *catarrhal* type, but the *follicular*, *œdematous*, and *suppurative* types of pharyngitis are rarely met with, unless in conjunction with more or less severe involvement of the tonsils.

Etiology.—Acute tonsillitis is supposed still by some observers to be a manifestation of the rheumatic diathesis, but the trend of opinion is in the direction of regarding *every* case of tonsillitis, whether accompanied by signs of general pharyngitis or not, to be of septic origin. The nature of the infection is already discussed upon page 1287, and, as stated, probably there is no specific micro-organism, the infection being usually multiple, or consisting in some cases mainly of staphylococci, pneumococci, or streptococci, the latter being found usually in the more severe forms,

The disease in probably all its varieties is distinctly contagious, and the microbes are conveyed in the secretions from the throat, usually in the spray produced by coughing; but they may be carried in the air, as in sewer-emanations, or conveyed in milk or water. It is not rarely associated with influenza, scarlatina, measles, and other exanthemata. The presence of a previous hypertrophic condition of the tonsils apparently renders the individual more susceptible to the infection, and this susceptibility in all cases apparently increases instead of diminishing after repeated attacks, till the individual passes middle-life.

The forms or varieties of acute tonsillitis are numerous and frequently merge into each other; whether they are each the result of a different microbe is not easily demonstrable, since there is obvious difficulty in determining the prevailing organism in a multiple infection. Usually the following varieties are recognised—viz. the *catarrhal* or *erythematous*, the *lacunar* or *follicular*, the *suppurative* or *parenchymatous*, the *ulcerative*, and the *peritonsillar*.

Catarrhal and lacunar tonsillitis are the forms usually met with in every case of pharyngitis which is acute and severe. These latter varieties need not be described, as they are embraced in the description of acute pharyngitis upon page 1287. The lacunar form is of importance, since it is not infrequently mistaken for diphtheria, but a careful examination of the throat will reveal the localisation of the exudate to be confined to the crypts or follicles. It consists of a yellowish-white pultaceous semi-membranous secretion which as a rule is easily brushed off, as it does not usually blend or fuse with the similar patch over the neighbouring crypt. There is no bleeding or excoriation left after the removal of such an exudate, and the bacteriological examination never reveals the Klebs-Loeffler bacillus.

Acute ulcerative tonsillitis is a rare form of the disease, only seen in weak debilitated subjects, and then usually in company with some form of stomatitis, as the mercurial. The ulcers are multiple and well-defined, and the condition must be recognised as entirely different from follicular tonsillitis, which is often, though erroneously, styled '*ulcerated sore-throat*,' because the specks of creamy exudate are commonly mistaken for ulcers.

Peritonsillitis, like parenchymatous tonsillitis, is very liable to end in suppuration. In this form the inflammatory action, also the result of septic invasion, is localised in the loose fibrous tissue outside the tonsil. This variety probably always is the result of an extension outwards of the last-named condition or of the parenchymatous form; it may end in deep suppuration, which may expose the internal carotid and invade the deep cellular tissue of the neck.

There remains but the severe form of *suppurative* or *parenchymatous* tonsillitis to be considered. This is known by a variety of names, the most common being that of *Quinsy* or *Cynanche Tonsillaris*, though these names are also sometimes applied to the other varieties when they appear in an unusually severe form.

In this variety of tonsillitis the etiological factors correspond in the main with those of the other forms, but the predominant infecting organism is probably always streptococcal. The attack not infrequently follows exposure to cold, and in some instances direct infection from a similarly affected patient may be traced. This latter occurrence is common in those cases of acute pharyngitis recognised as hospital sore-throat, which sometimes pass rapidly into suppuration of the tonsils.

Symptoms.—In severe cases the invasion is abrupt, commencing with a rigor, high pyrexia, intense prostration, joint and muscular pains, headache, and backache. The clinical picture, omitting the throat-symptoms, is practically identical in a typical case with that observed in a severe attack of true influenza, both being due to a profound toxæmia. The throat-symptoms may immediately precede, accompany, or rapidly follow the first feeling of severe illness. Usually the first warning received by the patient is a pain observed in swallowing the saliva, as if a knife-blade had found its way into one tonsil; there is a severe aching or throbbing felt in the gland between the intervals of involuntary swallowing, and the entire throat feels dry. The glands about the angle of the jaw swell rapidly, and there is very marked tenderness to pressure applied by the finger from the outside. Very soon the lower jaw stiffens so that the mouth cannot be opened, and by the time the physician first visits the patient he may, especially in the case of very young subjects, be unable to get a satisfactory view of the interior of the throat. The saliva increases, and in the agonising attempts to swallow it the pain shoots up into the ear, and in the attempts to hawk up the mucous secretion from the throat intense pain is also experienced. The voice becomes nasal and speaking is difficult; occasionally aphonia is present. The breathing is, however, seldom seriously affected unless in rare acute cases where both tonsils are inflamed simultaneously and intensely. Upon inspection the interior of the throat is found deeply congested; one tonsil is usually much more swollen than the other, and frequently to a degree which causes marked bulging of the livid or scarlet-coloured mucous surface of the soft palate, which projects forwards into the buccal cavity. The surfaces of both tonsils are not infrequently covered over with patches of the thick creamy or pul-taceous secretion observed in the follicular variety of

the disease, since the inflammatory action, though starting deeply in the parenchyma of the gland, soon invades its entire structure. When both glands are involved simultaneously they may meet in the middle line, pushing forwards the oedematous uvula, which may appear as thick as the finger and blocking up the entire pharynx, so that, in attempting to swallow, liquids may come back through the nose. More frequently the other tonsil becomes markedly swollen after a variable interval, usually, however, not till suppuration or more or less resolution has taken place in the one first affected. Rarely do both proceed finally to suppuration. The secretions of the mouth and the breath become usually highly offensive, the tongue is coated deeply, and the salivary glands swell. As the fever continues, with the pain, rapid pulse, and other smart constitutional symptoms, a mild degree of delirium not unusually accompanies the severe restlessness, and a roseolar septic rash is not uncommon, which often raises a doubt about the presence of scarlatina.

The advent of suppuration about the fourth day is sometimes heralded by a rigor and intensification of the throbbing pain; about this stage, if the finger be introduced into the mouth, fluctuation may be detected, usually in the region of the soft palate, and occasionally this may be elicited by palpation from the outside, behind the angle of the jaw. By the bimanual method the presence of pus may be more certainly demonstrated. If the case be left to itself the abscess generally bursts into the mouth in the act of swallowing the saliva, and its evacuation is suddenly followed by intense relief. In one instance only, in the writer's knowledge, did suffocation occur from rupture into the larynx; but a fatal hæmorrhage has been known to result from suppuration involving the internal carotid, though this is only likely to occur when peritonsillar inflammation supervenes.

The progress of a severe case seldom extends beyond a week, and the patient is left in an exhausted condition by the severity of the fever and the enforced starvation caused by the pain of swallowing. In rare cases the second tonsil takes on acute parenchymatous inflammatory action several days after the attack has apparently passed off, in which case the illness may be protracted beyond the second or third week. Suppuration only occurs in the severer cases; gangrene is a rare event. Many mild examples of parenchymatous tonsillitis resolve without pus-formation, though in some of these probably small superficial abscesses may discharge themselves without being recognised. A not infrequent ultimate result is a permanent enlargement of the tonsil, especially after two or more acute attacks.

The form of acute tonsillitis recognised as *Vincent's Angina* is usually found associated with the cutting of either set of

teeth, and is due to the fusiform bacillus and the spirillum, which, acting together, produce the ulcero-membranous variety of the affection, not infrequently ending in extensive ulceration of the palate, tonsils, and fauces.

Diagnosis.—Usually there is little difficulty when the physician can obtain a satisfactory view of the pharynx. In the early stage *scarlatina* and *diphtheria* may be eliminated by a study of the history and invasion. In scarlatina of the anginose type the rash is usually delayed, and often not well-marked when it does appear, but generally the symptoms are of a distinct adynamic type from the first, and the throat-signs are characterised by a uniformly diffused lividity which involves the entire mouth and pharynx, spreading upwards into the nasal region, causing ichorous discharge from the nostrils, and downwards towards the larynx, interfering markedly with the respiration; and the septic process soon tends to extensively invade the glands and deep cellular tissue of the neck. Vomiting is usually a prominent feature, and the entire clinical picture is one of a more profound sepsis than is to be met with in tonsillitis. The presence of the Klebs-Loeffler bacillus must be relied upon, in all cases when detected, in establishing the diagnosis of diphtheria, though failure in demonstrating its presence is not equally conclusive in the elimination of the disease.

Prognosis.—This is always favourable, except in the very rare instances where there is great œdema or where the suppurative or necrosing process involves a large vessel. As a rule, even when the symptoms appear formidable, the patient may be safely assured that there is no danger of suffocation, since tonsillitis, unlike the scarlatinal condition, shows no tendency to extend towards the larynx.

Treatment.—The general indications are those of acute pharyngitis, and local measures are of more value than internal remedies. Gargles are not to be relied upon, owing to the pain and difficulty in their use; but a weak carbolic spray is at all times beneficial, since it combats sepsis, and by its local anæsthetic action it markedly minimises the attempts at swallowing and hawking up the tenacious secretions. Nothing, however, is so soothing as the vapour inhaled from boiling water by holding the mouth over a basin of the fluid, with a large towel or small sheet loosely thrown over the patient's head. Scarification of the tissues or multiple punctures should be made by the point of a sharp guarded bistoury when there is much œdema present, even when no suspicion of pus exists. As soon as distinct fluctuation can be made out, the point of a guarded or sheathed bistoury should be inserted for the depth of half or three-quarters of an inch into the softened area at its upper and outer aspect, the knife being withdrawn in the opposite direction, cocaine

having been previously swabbed over the inflamed tonsil. St. Clair Thomson effects the evacuation of the pus by means of a Liston's dressing forceps with a sharp end, which removes all danger of injury to the carotid artery.

CHRONIC ENLARGEMENT OF THE TONSILS

Etiology.—A very common cause of permanent enlargement of the tonsils is one or more preceding attacks of acute inflammation. After each attack of sore-throat the organs return more and more slowly to a condition approaching the normal, till finally they remain in a state of apparent hypertrophy. This result is liable to follow all forms of pharyngeal or tonsillar inflammation. It sometimes supervenes after scarlatina, measles, whooping-cough and diphtheria, and is not very uncommonly found in association with rickets. In but a very small percentage of cases the enlargement or hypertrophy seems to be of congenital origin; in a not larger percentage of cases it is one of the local manifestations of the status lymphaticus, in which there is hyperplasia of the lymphatic glands and of the lymphoid tissue throughout the intestine, spleen, thymus, thyroid and bone-marrow. It does not appear that the condition should be regarded as really of a tubercular nature, though true tubercle of the tonsils occasionally is seen; nevertheless, in the scrofulous diathesis it is common to find the tonsils more or less hypertrophied. In a large percentage of the cases commonly met with in daily practice there is no assignable cause for the hypertrophic enlargement; the writer has, however, so frequently found the condition in association with caries of the teeth that he is satisfied the accompanying septic condition of the mouth often is the starting-point of the tonsillar hyperplasia. Vincent emphasises the frequency with which his angina, especially in the ulcero-membranous form, is found in association with carious teeth. The condition of the dentition should always receive attention in every tonsillar lesion. (The question of the invasion of the system by the bacillus of tubercle *via* the tonsil has been already discussed under the etiology of phthisis.)

Pathological Anatomy.—The swollen organs may show a uniform hypertrophic enlargement in which all the tissues entering into their composition may be the seat of increased growth. More frequently, however, the hyperplasia predominates in the *lymphoid* elements, in which cases adenoids are also usually present. Either at a much later stage, or arising as an independent condition from the first, the *fibroid* form of the affection is seen, in which the fibrous tissue is extensively proliferated, forming a tumour of dense hardness which may cut like cartilage. All three forms of tonsillar enlargement may be associated or complicated with foul

smelling solid green pellets of secretion retained in the lacunæ of the organ. Sometimes this form of *chronic lacunar tonsillitis* exists without any hypertrophic enlargement; it may be even met with where the organs are diminished in bulk, and occasionally the retention of the secretion leads to the formation of calculi or tonsilloliths or to cystic degeneration, but in a considerable proportion of the cases of tonsillar calculi the calcareous material originates in the invasion of the crypts by the *leptothrix buccalis*, which is always present in the tartar around the teeth.

Tubercle bacilli are found in a very considerable proportion of the cases of enlarged tonsils, and especially where the cervical glands are also enlarged, but as yet it is impossible to say how far the invasion is a secondary event.

ADENOID GROWTHS

Since attention was first called by Czermak and Myer to the condition in which adenoid tissue is found in the nasopharynx, the etiology and the ultimate evil effects of the vegetations have received considerable study.

Pathology.—These adenoid growths result from a hyperplasia of the mass of lymphoid tissue known as Luschka's tonsil, normally present in the post-nasal space in young subjects. The lymphoid tissue is attached to the vault of the naso-pharynx, and may extend to the sides of Rosenmüller's fossa and involve the orifices of the Eustachian tubes. It forms lobed or grooved masses, pedunculated or sessile, which, in addition to the ordinary soft lymphoid tissue, contains a variable amount of fibroid material; hence some are soft and vascular, resembling nasal polypi, and hang down like papillomata from the vault, whilst others, like sessile granulations, are seen on the pharyngeal wall. The fibroid varieties are firm and oftener sessile, forming smooth tumours of ill-defined shape. They are covered by two or more layers of epithelium, the most superficial of which is often ciliated, and retention-cysts are commonly observed in sections of the tumours. Tubercle bacilli have been found by different observers in a variable proportion of adenoid growths—from 3 to 16 per cent.; and staphylococci and streptococci are present in more than half the cases. The relationship of adenoids to tubercle is a subject of great importance; all that need be here said, however, is that the bacilli are usually found lying immediately under the epithelial lining, especially in the vicinity of the crypts, so that it would appear, according to Lack, that the adenoids may offer an entrance for tuberculous infection. That this is more than probable seems a safe inference from the almost constantly present chain of small hard glands, palpable along the posterior edge of the sternomastoid muscle, though this last-mentioned authority

attributes the latter phenomenon to the infection of the cervical glands by the other organisms present.

Many of these vegetations appear to wither up and disappear later on in life, but during their existence they lead to serious interference with breathing and development.

Etiology.—Adenoids are very frequently associated with the chronic hypertrophic changes already described in enlargement of the tonsils, in which cases they are the outcome of the same etiological factors as attacks of sore-throat, diphtheria, measles, scarlatina, whooping-cough, and nasal catarrh or lymphatism.

Age is an important factor, most of the cases being observed in children under ten years; they have been seen soon after birth, but are probably seldom congenital. Heredity plays a part in many instances, several members of the same family being found to suffer from adenoids during childhood. As the lymphoid tissue usually begins to atrophy between the ages of fifteen and twenty, their presence is rare after this period, but in neglected cases operative interference has been necessitated in subjects even past middle-life.

Great diversity of opinion exists about the prevalence of these growths, some authorities placing the percentage of adenoids as high as nearly 50 per cent. in all ill-nourished children. The disease is certainly much more common in cold damp climates. Some authorities consider that all inherited malformations of the nasal passages lead to the formation of adenoid growths, but Lack very properly regards the adenoids as the cause of these structural peculiarities. As regards the question of tuberculosis being an etiological factor, the fact already mentioned appears to place this agent also in the category of a result and not of a cause.

Symptoms.—These are similar to the local disturbances and obstruction which result from chronic enlargement of the tonsils, and, as both conditions often co-exist, they may be considered together, always remembering, however, that the most serious obstruction may originate from adenoid vegetations, which sometimes exist without any obvious tonsillar enlargement.

The chief mischief caused by these conditions arises from the chronic nasal obstruction which they produce interfering with the development of the thorax and lungs, and with the growth of the entire organism.

The physiognomy of the condition when well-marked is usually highly characteristic: the child keeps his mouth open, the lower jaw hangs loose, the upper lip is shortened, and the normal folds around the mouth are absent. The nose is altered in shape: its bridge appears wide or flattened, the alæ nasi are collapsed, causing the apertures of the nostrils to be contracted, and there is a deep groove where the cartilages

and the nasal bones join. With these anatomical changes there is always associated a dull, listless, stupid appearance, which may, by the absence of intelligent expression, amount to a semblance almost of idiocy. There is usually more or less deafness present along with the apathy and mental backwardness which accentuates the stupidity of the child.

There are well-marked changes in both jaws, which remain long ill-developed: the alveolar arches are V-shaped and the roof of the mouth high and narrowed; the teeth are placed irregularly, owing to their being crowded together in the stunted jaws, the central incisors being sometimes placed edge-wise and overlapping each other, and in the upper jaw they protrude from under the short thick lip. Speech peculiarities are always well-marked; the voice-sounds resemble those which in health can be produced by pinching the nose between the thumb and fingers before attempting to speak. The letters 'm' and 'n' are sounded like 'b' and 'd,' as if the patient had a severe catarrh of the nose—indeed, catarrh of the nose is a very frequently recurring result of the obstruction in most cases, and the sense of smell is impaired; stammering or stuttering is often present, though not due directly to the anatomical obstruction.

Mouth-breathing is constantly present, either partial or complete; snoring at night is almost invariably produced. Even during the waking hours, especially upon exertion, some snorting sound often accompanies the respiratory acts. During sleep the respiration is disturbed in various ways, because usually the child attempts to inspire through the obstructed nasal passages; the *alæ nasi* sink in, and the narrowed chinks of nostrils permit of no air entering through the obstructed naso-pharynx, so that the thoracic walls collapse, and the child wakes suddenly, with a distressing sense of suffocation, which is only relieved by deep voluntary inspirations through the mouth. It is certain that in sleep, though the child's mouth is wide open, little or practically no air passes through the mouth at all, since the tongue falls back and entirely obstructs the passage of the air through the fauces. These fits of irregular and disturbed breathing come on repeatedly through the night; they break the sleep and lead to distressing dreams or night-terrors. Cough is also usually present at night, and there is cyanosis or lividity and profuse sweating from the imperfect oxygenation of the blood; the pulse fluctuates like the respirations, and quickens markedly during the struggle for breath, which usually occurs immediately before awaking.

These embarrassments in the respiration lead to grave changes in the shape of the thorax, causing often permanent deformities. The typical chicken or pigeon breast may ensue, as described upon page 1525.

The mechanism of this deformity may be readily understood by watching the sleeping child. Each forced inspiration, failing to draw air through the obstructed naso-pharynx into the lungs, causes recession of the anterior triangles of the neck, and of the fossæ above the sternum and clavicles, whilst the lower ribs and inferior end of the sternum also fall in or recede. As pointed out by Lack, if the bones are strong there will probably be a depression in the lower sternal region and round the lower part of the thorax, corresponding to the attachments of the diaphragm; if they are soft, as in rickets, which is not an unusual complication, the sides will be flattened, the pigeon-shaped deformity being produced. Occasionally, when asthmatic seizures accompany adenoids, the shape of the thorax approaches to that of the barrel-chest, typical of chronic emphysema. It is probable, also, that the so-called 'funnel breast,' which is characterised by a deep hollow at the bottom of the sternum, may be the result of adenoids. In all these permanent deformities the adenoid vegetations play a far more important rôle than the chronic enlargements of the tonsils, but obviously the condition is aggravated when both exist together, as they so frequently do.

Coincident with these anatomical characteristics of the disease produced by the mechanical obstruction in the naso-pharynx is the mental condition already incidentally referred to in the description of the facial peculiarity. The intellectual faculties do not develop as in healthy children; stupidity, more or less marked, is always present, and may be detected by the child's difficulty and slowness in answering ordinary questions and in not taking notice of the trivial passing phenomena which usually attract children's attention; they learn simple lessons with difficulty and are easily tired in any mental effort, and are often pœvish or morose in disposition. To this group of nervous or mental symptoms Guye has given the name of *aprosexia*, and he believes that they are caused by the interference of the lymphatic outflow from the brain caused by the nasal obstruction. The general bodily development usually keeps pace with the mental backwardness, and such children are easily tired by moderate exercises, are puny and always ailing, and usually more or less anæmic. The stunting of the growth has been conjectured to be the result of the connection between the functions of the pituitary body and the tonsils in embryonic or infant life; more probably it is chiefly due to the chronic interference with the proper aëration of the blood, since it rapidly passes off as soon as the naso-pharynx is cleared of the obstructing growths. Other and remote effects are usually present, probably of reflex origin, such as nocturnal enuresis, stammering, and stuttering, epileptiform convulsions during sleep, laryngeal spasm,

spasmodic cough, asthma, headache, and habit-spasm of the facial muscles.

Subjects suffering from adenoids are constantly liable to attacks of catarrh of the nasal passages and of the pharynx; recurring attacks of sore-throat, either pharyngeal or tonsillar, are constant symptoms, so that rhinitis, catarrhal or hypertrophic, is almost as constant as tonsillar hypertrophy. Deafness, usually preceded by recurring attacks of earache, as already mentioned, is very common, and so is epistaxis; the breath is often highly offensive, either from the decaying teeth, which necrose early, or from the evil-smelling pellets of offensive secretion usually present in the follicular recesses of the tonsils. The senses of smell and taste are always blunted; often the former is entirely absent.

Diagnosis.—There is usually no difficulty in the detection of the condition by a simple inspection of the physiognomy alone. The history of the case is equally distinctive: the loud snoring and snorting during sleep, with long pauses, followed by noisy breathing as the child partially or completely wakes up, leave little doubt about the cause. The use of the laryngeal mirror reveals the growths *in situ*, or these may be felt by the physician's forefinger introduced through the mouth. The chain of small indurated glands along the posterior margin of the sternomastoid, when found in association with chronic enlargement of the tonsils in a child is almost pathognomonic.

Prognosis.—In the very mild cases where, though the growths are known to exist, no symptoms or signs of obstruction being present, the prognosis may be hopeful that the condition will entirely pass off after puberty without surgical treatment, as the lymphoid tissue tends to commence withering up before twenty. Much more frequently, however, the problem in prognosis is a very different one, and usually resolves itself into the question of, to what extent the anatomical, mental, and other changes can be expected to clear off after operation. As a rule, in all save long-neglected cases, the outlook is very good, the most striking improvement setting in rapidly after extirpation of the growths; even the hearing commences to improve almost immediately, and unless destructive middle-ear lesions have occurred it usually becomes normal. The chest-malformations, unless in neglected cases where the bones have long set in their abnormal positions, speedily begin to improve if care be taken that proper breathing-exercises be persevered with. The mouth, however, usually alters little, the arch of the palate remaining often high and narrow and the anterior part of the dental arch crowded; the nasal septum may remain deflected or distorted as the floor of the nasal cavity is raised, causing a diminution in the vertical diameter of the nose.

Stammering or stuttering and nocturnal enuresis do not pass off as a rule like the other symptoms, but generally asthmatic seizures disappear entirely or show great amelioration.

Recurrences after removal are so rare that a satisfactory prognosis in this respect may be confidently made, though occasionally repeated attacks of rhinitis may have caused changes in the inferior turbinates which may demand the removal of their posterior extremities at a later stage.

Treatment.—*Enlargement* of the tonsils, when producing the symptoms of obstruction as above described, should be always removed by the guillotine, by the galvano-cautery, by morcellement, by the ccraseur, or by enucleation, as by the time obstructive signs have become established little need be expected from drugs, local astringents, or caustics. The moderately enlarged tonsils of adults may sometimes be caused to shrivel up by repeated puncturing with the fine point of the galvano-cautery (ignipuncture).

Adenoids in like way should be surgically treated in a radical manner as soon as their presence produces signs of nasal obstruction. Mild cases, where the size of the vegetations do not interfere with breathing, may be treated by improved hygiene, iodides, breathing exercises, and removal to a drier climate; but as a rule, as soon as advice is sought the case requires prompt surgical treatment. The operation may sometimes be easily performed under local anæsthesia, and when chloroform or other general anæsthetic is necessary great care should be exercised lest asphyxia result from the blood entering the larynx. It is therefore advisable not to permit the anæsthesia to proceed to the abolition of the swallowing and cough reflexes. Chloroform is held by many operators to be especially dangerous, and nitrous oxide or ethyl chloride is usually preferred; but the A.C.E. mixture is convenient and safe, if the anæsthesia is not permitted to proceed to the profound or complete degree of total insensibility. The removal was formerly effected by the finger-nail; now Gottstein's curette is employed; Löwenberg's or Juracz's forceps are preferred by most operators. The dangers of hæmorrhage are, upon the whole, slight, even when both tonsils and all the adenoid vegetations are carried out at one sitting, which is the most desirable procedure.

TORTICOLLIS, or SPASMODIC WRY-NECK

The muscular rheumatism or fibrositis which produces lumbago very often manifests itself in so-called wry-neck, or *stiff-neck*, which is associated with some deviation of the head, tenderness of the neck-muscles, and great pain on

movement, obviously the result of a rheumatic fibrositis affecting the sheaths and fibrous tissue of the cervical muscles after exposure to cold.

Another variety of wry-neck is known as *congenital* or *fixed torticollis*, in which, owing to structural shortening of the sternomastoid muscle, commonly the result of injury during delivery, the face is deviated to one side and cannot be turned towards the side upon which the muscle is shortened. The muscle affected is the right sternomastoid in the great majority of cases, and this has led to the belief that often no trauma is answerable for the fixation, which is then due to diminished growth of the muscle in its length, owing to the position of the head in the pelvis before birth. By some authorities the shortening is supposed to be due to some developmental error in the nuclei of the medullary nerves, as often facial asymmetry accompanies this congenital variety; but there is no element of active spasm in fixed torticollis, and it always disappears upon section of the tendon of the shortened muscle.

True spasmodic torticollis—the condition under consideration—is a totally different affection, being of the nature of a neurosis, arising independently of any morbid condition of the muscles.

Etiology and Pathology.—The disease is most common about middle-life, and women are affected more than twice as frequently as men; the affection is rare in childhood and old age. Many of the cases in young women are associated with hysteria, and a history of inherited neurotic tendency is not rare in either sex, epilepsy, asthma, megrim, or other allied neuroses or habit-spasm being sometimes present in the sufferers or in their near relatives.

As exciting causes, profound mental emotions, shock, traumata, exposure to cold, excessive use of the neck-muscles, and severe neuralgia have been occasionally recognised; but in the majority of cases no apparent cause can be detected, the spasm slowly setting in during a condition of obvious robust health.

The pathogeny of the condition can only be surmised, as in the case of epilepsy and other purely functional affections which exist without recognisable organic lesions. It is considered to be due to the over-action of the nerve-cells in the higher cortical or lower spinal centres, or in both, but the causes which induce this instability are unknown. The muscles mainly involved are those supplied by the spinal accessory nerve, but those receiving their supply from the upper spinal nerves also suffer more or less.

Symptoms.—The onset of the spasm is as a rule insidious, requiring many months for its typical development, and occasionally it is preceded by marked stiffness and severe

neuralgic pains ; in rare cases the spasm appears suddenly, and the fully developed affection may be recognised within a day or two after the first appearance of anything being amiss with the neck.

The spasm affects usually the sternomastoid first and often most severely throughout, but upon the whole it is seldom that this muscle alone remains involved, the superficial and deep muscles of the neck being generally affected, more or less, on both sides, though usually more upon one side than the other. The upper part of the trapezius on the same side suffers next in frequency with the opposite splenius, the scaleni suffering usually less frequently. The spasm may be either tonic or clonic, and often both types of spasm may be present in the same patient at the same time. In the rare cases where the spasms are equally severe upon each side of the neck, the head is carried directly backwards by the strong action of the splenii, so that the face may be horizontal, looking upwards towards the sky, the sternomastoids intensifying the retroflexion. This variety is known as *retrocollic spasm*, and is always accompanied by an associated movement of the frontales muscles which elevates the eyebrows and which definitely excludes all forms of simple tremor.

Since the spasm, though commonly bilateral, is nearly always greater upon one side than upon the other, it follows that there is deviation or rotation of the head in nearly every case, and the degree and variety of this depend upon the combination of muscles involved in the spasm. When the sternomastoid alone is involved the *face* is turned towards the opposite side ; the *head* is rotated and slightly inclined towards the affected side. Gowers states that the common combination of the sternomastoid with the upper portion of the trapezius upon the same side greatly increases the inclination of the head towards the shoulder, which prevents any increase in rotation. When the sternomastoid is involved with the opposite splenius there is extreme rotation, so that the face is turned towards the shoulder upon the side of the affected sternomastoid.

When the spasms are tonic and increase in severity they tend to become clonic, which causes jerky movements. When remaining tonic they are seldom absent during sleep in severe cases ; the clonic spasms as a rule disappear during sleep. Though the spasms usually vary in their intensity from time to time, the muscles are rarely free from contraction for more than a few minutes at a time. As the disease progresses the spasms tend to spread and involve other than the neck-muscles : thus the shoulder girdle, arm, and face muscles may suffer, and these sometimes may be thrown into action by a forcible attempt to keep the head in the normal position. Mental excitement, muscular fatigue, and sometimes a mental

effort made to control the spasms, increase the severity of the contractions. There is usually but little cramp-like pain accompanying the spasms, but sometimes, especially when the arms are affected, this is very severe; commonly there is considerable dull aching pain and a distressing sensation of fatigue in the contracting muscles, occasionally associated with tingling due to compression of the nerve-trunks from the rigid tetanic contractions; severe neuralgic pain, except at the onset, is rare.

The muscles are never observed to waste; often some hypertrophic enlargement may be detected in those most constantly the seat of spasm in chronic cases. Their electrical excitability is sometimes normal, but usually it is considerably exalted.

The disease is generally a chronic one, in which remissions and exacerbations often alternate with each other. In many cases which apparently are of a severely progressive type up to a certain point, the disease may gradually or suddenly assume a permanently stationary character for the remainder of life. Complete resolution sometimes takes place, but recurrences are very liable to supervene after some severe muscular fatigue, exposure, or acute mental excitement, so that the disease must be regarded as one of the most obstinate of all the neuroses.

Diagnosis.—Once the neurosis has become established there is practically no difficulty in its recognition. From the so-called '*wry-neck or rheumatic torticollis*' which directly follows exposure to cold the disease is recognised by its insidious onset, marked deviation or rotation of the head coming on in frequent paroxysms of true spasm, and there is practically no tenderness in the muscles, which latter is a constant feature in rheumatic stiff-neck.

False torticollis must be excluded; this is a condition in which deviation of the head occurs independently of spasm or muscular contraction, and is usually caused by disease of the cervical vertebræ. The sternomastoid is put upon the stretch (but is not in a state of spasm) on the side towards which the face is turned; whilst in spasmodic torticollis the face is turned in the opposite direction to that in which the contracted muscle is situated.

Hysterical spasm may be recognised by its extension to the trunk and to other muscles never involved in torticollis. The stigmata of the hysterical condition are always found to be present when carefully looked for; but true torticollis may co-exist with hysteria, in which case the spasm may be confined to the neck-muscles. Spasms affecting the neck-muscles which originate in *cortical cerebral disease* and *pontine lesions* are usually associated with conjugate deviation of the eyeballs.

Prognosis.—This has been dealt with in the remarks made upon the progress and duration of the disease. There is no tendency towards a shortening of the patient's life, but the prognosis should be always very serious owing to the intense discomfort and sometimes severe suffering and incapacity which it inflicts. In a given case the severity and long duration of the spasms justify the forecast of an incurable disease, unless radical surgical measures be resorted to.

Treatment.—Nervine sedatives, as morphine, hyosine, atropine, chloral, conium, gelsemium, &c., control the spasms, but are practically inadmissible owing to the chronic progressive nature of the malady. Electricity, counter-irritation, galvanism, section of the muscles or of their tendons, and fixation of the head have been frequently resorted to, and always result in failure. The only remedy of any real and lasting value is the excision or resection of a piece of the spinal accessory or other nerve supplying the affected muscles; even this sometimes fails and may necessitate a further neur-ectomy of the posterior branches of the spinal nerves supplying the splenii. In mild cases nerve-stretching has occasionally proved beneficial, but the spasms as a rule return after a longer or shorter interval.

In fixed wry-neck the division of the tendon of the affected muscle is usually followed by satisfactory and permanent results.

TRANCE. (See under HYSTERIA, p. 692.)

TREMORS. (See under SPASMS, p. 1620.)

TRICHINIASIS

This is a disease chiefly involving the muscular system, caused by the presence of the encysted larvæ of *Trichina spiralis*.

Etiology.—The disease is rare in Great Britain, but common in North Germany, Holland, and Denmark, where it occasionally appears in almost epidemic form, though the close attention paid to the inspection of food in Germany has considerably lessened its prevalence. It is occasionally found in all regions where the meat prepared from pigs is used as food. The parasites gain their admission to the human body in every case through the eating of affected pork, ham, bacon, or sausage which has been imperfectly cooked or eaten raw. The life-history of the parasite has been studied by many observers, and Manson accepts the view that the common

grey rat is its normal host, pigs probably becoming infected by eating dead rats or the offal from slaughter-houses. The encysted larvæ in the muscles of the pig, after gaining entrance to the human stomach, have their envelopes digested by the action of the gastric juice; the liberated embryos pass into the small intestine, where their sexual development is perfected in twenty-four to forty-eight hours. The mature male worm measures 1.5 mm., the ovoviviparous impregnated females, which are much more numerous, are almost twice as long. The smaller worms are recognised as males by their possession of two minute prehensile organs situated at the hind-end of their cylindrico-conical bodies. After copulation the male worm dies and the impregnated females increase rapidly to their full dimensions, pouring into the intestine many hundreds of living embryos as early as a week after gaining entrance into the human subject, and these embryos continue to be passed by the parent-worm for more than a month later, till her death occurs.

The young free trichinæ penetrate the intestinal walls, and finally, within a period of seven or eight days, reach the interstices between the fibres of the striped muscles of the different parts of the body, where, owing to the irritation which they cause, an oval cyst develops of about the length of half a mm., its long diameter lying in the direction of the muscular fibres. Inside this minute chitinous capsule or cyst the trichina lies coiled up and surrounded by a clear albuminous fluid. Several small trichinæ may be seen in a single cyst; more frequently, however, only one is found in each. At this stage of its existence it appears as a small cylindrical worm of about 1 mm. in length, with its hinder extremity more or less bluntly conical, the fine filiform end terminating in a mouth. Many of the young trichinæ appear to penetrate the sarcolemma and gain the interior of the muscular fibre. Within a period of three weeks the encapsulation may be complete, but usually the cysts are not to be detected till about five weeks after the swallowing of the trichinised food.

The capsules are most numerous in man in the intercostals, the tongue, eye, and neck muscles, diaphragm, loin, laryngeal and facial muscles; but all the muscles of the body may be affected. In the cystic stage the further growth of the parasite is arrested, and in this condition it remains motionless and passive, but still capable of further development on being transferred to the stomach of an animal; this latent period has been proved in some instances to be as long as a quarter of a century. In most cases of trichiniasis in man, however, the capsule becomes the seat of calcareous changes, and the parasites die usually within a period of six months from the obliteration of the cysts by the deposition of lime-salts, in which case they become opaque, hard, and gritty, and are

frequently detected in the dissecting-room by the grating of the scalpel in the cleaning of a muscle. In the muscles of the pig calcification is rarely ever witnessed, probably owing to the shortness of the life which the animal is permitted to enjoy, so that the cysts are only to be detected after careful examination.

In the encysted stage the parasites exhibit remarkable resistance to changes of temperature: they withstand cold many degrees below the freezing-point of water for long periods; they are practically unaffected by the pickling, smoking, and curing processes usually employed in converting pork into ham and bacon. A temperature of 80° C.—*i.e.* 176° F.—is necessary for their destruction, and this degree of heat is never reached in the centre of a large ham until the boiling has been continued for many hours. Thus Manson states that the temperature of a large ham at its centre is only 65° C. after six hours' boiling; he attributes the rarity of the disease in Britain to the thorough and prolonged methods of cooking.

Symptoms.—Trichinosis or trichiniasis in man usually only becomes recognisable when a considerable number of the encysted parasites have escaped destruction in the imperfect cooking process to which the trichinised food has been subjected. Hence the worst cases are commonly seen after the cured or smoked flesh of the pig has been eaten raw, as it often is in North Germany.

The symptoms differ with the developmental stages of the parasite. After the introduction of trichinised meat into the human stomach there is a period of several hours in which no evidence of the infection is forthcoming, but before the end of a complete day, and often within six or eight hours, serious symptoms of gastro-enteric irritation or inflammation, as vomiting and purging, with severe colic, set in, so that the case may be mistaken for cholera or for some form of irritant-poisoning. In some epidemics these intestinal and gastric symptoms may be entirely absent, though the subsequent progress of the disease may be towards a fatal issue. As in other forms of poisoning, the early intensity of the vomiting and purging may clear the alimentary tract of the greater portion of the irritating material, so that some of those who had partaken of portions of the same joint suffer less seriously afterwards than individuals in whom vomiting and purging were absent.

Where the gastro-intestinal symptoms are latent more or less languor, debility, and depression, with vague muscular or joint pains, may be complained of.

The next stage—that of the invasion of the muscles by the wandering young trichinæ—is manifested by the presence of more or less smart myositis about eight to ten days after

the ingestion of the encysted parasites. In some cases the initial intestinal irritation lingers till the advent of the muscle-trouble. Fever of a remittent type is usually present, the evening temperature reaching 102° , or even 104° , with a drop of a degree or a degree and a-half in the morning; sometimes there are complete intermissions and occasionally shiverings, and the pulse is much accelerated. The muscles become tender to pressure, as elicited by the touch and by every effort to bring them into action; hence the limbs are kept instinctively by the patient in a state which permits of the greatest degree of muscular relaxation—*i.e.* in a condition of semi-flexion. Some spontaneous muscular pain is always present, and the intensification of this upon movement is highly characteristic. A marked rigidity and swelling of the muscles are present early, and a very considerable degree of œdema of the skin covering them occurs later on in all severe cases; but a characteristic anasarca appears in the face for a few days as soon as the first evidence of muscular involvement sets in, which gives a remarkable owl-like aspect to the patient owing to the swelling of the tissues around the orbits—a condition differing entirely from the œdema of renal disease.

Serious symptoms occur from the myositis attacking certain muscles. Thus dyspnœa or painful respiration may arise from the condition of the diaphragm and intercostal muscles; mastication may be rendered almost impossible by the involvement of the masseters; lingual, laryngeal, and pharyngeal difficulties render swallowing and phonation sometimes so painful that all efforts to chew, swallow, or speak are voluntarily suspended, and the patient may be unable to bring his ocular muscles into action. Profuse sweatings are common, and various urticarial, erythematous, or miliary eruptions may accompany the general œdema, and these are often associated with considerable pruritus. The patient wastes rapidly; bronchial or hypostatic pneumonic symptoms and signs sometimes show themselves owing to the difficulty in expelling the mucous secretion from the tubes. In severe cases delirium is common; this, with the fever, quick weak pulse, dry brown tongue, restlessness, insomnia, and alternating stupor, produces an almost typical typhoid condition.

The state of the blood has attracted considerable attention of late years; there is always marked leucocytosis. Osler emphasises the importance of observing the increase in the eosinophile-cells, first detected by T. R. Brown; these may number more than half of the total leucocyte count. Polyuria and albuminuria are not uncommon.

The progress and duration of the cases vary considerably with the intensity of the infection. In mild cases recovery may be complete in two or three weeks; in the more severe forms of trichinosis, however, the myositis usually shows

no signs of abatement till the fifth, sixth, or even the seventh week, or later. Convalescence is always slow, marked muscular weakness and anæmia remaining for months; and sometimes œdema persists for weeks after the subsidence of the myositis and pyrexia. When death occurs it is commonly about the fifth week; rarely has a fatal event been observed within the first ten days during the collapse resulting from the severe purging and vomiting. The most common cause of death is interference with the respiratory mechanism. The mortality in epidemics has varied from $1\frac{1}{2}$ to 33 per cent. The variations in the severity of the attacks occurring in individuals who have partaken of the same feast are probably due not so much to the amount of trichinised food eaten by each person, as to the parts of the joint from which they have been helped, since the trichinæ in the circumferential portions are usually destroyed completely, whilst those in the central parts are left intact. The attacks following the ingestion of raw, smoked, or cured ham or pork sausages are always, for obvious reasons, more dangerous than those arising from imperfectly cooked food.

Diagnosis.—Owing to the outbreak often following within twelve hours or less in a number of persons who have feasted together, the suspicion of *irritant-poisoning* by ptomaines or a mineral toxic impurity arises. A careful examination of the intestinal evacuations will set the suspicion at rest, since the parasites may be readily detected by the microscope at this stage, and the encysted trichinæ may be found in remnants of the food which have not been consumed.

Cholera, if epidemic or endemic, is excluded with difficulty, and Manson draws attention to the importance of observing the symptoms of pain and tension in the flexor muscles, which in trichinosis is often observed within the first three or four days before myositis supervenes.

Typhoid fever is excluded at this early stage by the suddenness of the invasion, by the absence of headache, by the intensity of the diarrhœa, by the investigation of the stools, and by the history of the case. At a later stage the myositis, leucocytosis and increase in the eosinophiles, the characteristic facial œdema, absence of rose-spots and of splenic enlargement leave no doubt about the diagnosis.

Acute rheumatism may be simulated by the intense pain and tenderness in the limbs, but these will be found upon examination to be localised in the muscles and not in the joints, since the latter are unaffected in trichinosis.

Henoch's purpura has been observed in two instances by the writer to closely simulate trichinosis; in both the characteristic œdema about the margin of the orbit was present, with intense abdominal pain and diarrhœa and smart pyrexia. The absence of the mature parasites in the stools settled

the question of diagnosis, though the severe pain in the limbs could not be demonstrated as existing only in the joints.

In all doubtful cases the muscles may be examined, a fragment of the biceps or pectoralis major being removed by means of Middeldorpf's harpoon. In the muscles of the human limbs the trichinæ are most commonly found near to their tendinous extremities.

In chronic cases, as pointed out by Manson, trichinosis closely simulates *beriberi*, since the following features are common to both affections: they occur in localised epidemics; pain and tenderness exist in the muscles; œdema and failure in respiratory power also are observed in both, but the symptoms in *beriberi* are those of a neuritis, as absence of knee-jerks, localised anæsthesia and reaction of degeneration, whilst in trichinosis the symptoms are those of a myositis following intestinal irritation.

Trichinosis may be mistaken for *acute Bright's disease*, only, however, when the general œdema is accompanied by marked albuminuria, which is a rare event in the affection; the œdema of the face is usually a transient phenomenon and occurs before the general œdema, which is always marked in the hands. It is worth remembering that even when the anasarca of trichinosis is very extensive the loose cellular tissue of the penis and scrotum generally entirely escapes the œdematous infiltration.

Prognosis.—This has been already indirectly dealt with in the remarks made upon the duration, course, and mortality of the disease, and little further need be said upon the subject, save that children almost always recover, and that the severity of the initial intestinal irritation bears no proportion to the subsequent myositis.

Treatment.—*Prophylaxis* is obvious. The disease is entirely prevented by thorough cooking of all food obtained from the flesh and tissues of the pig, and the latter animal would probably never become trichinised if exclusively fed upon grain, and if rats were prevented gaining an entrance into the sty. Where ham and sausages are eaten uncooked, as in North Germany, the pork is now submitted to rigid examination, a small portion of the tongue, diaphragm, intercostal and abdominal muscles being examined by the aid of a lens before the meat is submitted to the pickling, curing, or smoking process. The only danger in Britain arises in the case of very large hams which, if boiled sufficiently long to kill trichinæ in their central portion, are liable to be spoiled: hence such large joints should be always cut in two before cooking is commenced.

During the acute intestinal stage the diarrhœa should not be checked by opiates or astringents, but smart purgatives, as large doses of calomel, should be administered. All the drugs

employed for the destruction of the tæniadæ, cestoda, nematoda, and of anchylostoma have been tried ; these drugs, if of any value, can only act when the mature parasites are free in the intestines, as no agent possesses any influence over the encysted larvæ in the muscles. The most reliable anthelmintic is thymol in large doses. It must be remembered that the parasites, whilst remaining in the intestine, continue to shower their living young into the lumen of the bowel for at least four, or even six weeks, and in many instances after death the mature female worms are to be found in the intestine as late as this period. The pain, tenderness, and other symptoms must be relieved by appropriate treatment, and the patient's strength maintained by concentrated liquid nourishment administered at short intervals.

TRICHOCEPHALUS DISPAR

This is the name given to the slender nematode, better known as the Whip Worm, which is often found in the cæcum. Its presence as a rule causes no disturbance of any kind, though the parasite measures as much as 50 mm. in length, and may exist in large numbers. It is conveyed to the human stomach by the ova finding their way into the drinking-water ; and in many districts in the tropics and in some European cities, notably Paris and Kiel, a large percentage of the population are infested with the parasite. The only clinical importance to be attached to this worm is the recognition in the stools of its ova, which are smaller than those of the ascaris lumbricoides, and are distinguished by the minute aperture, which shows as a clear pale speck, situated at each end of the brown oval shell.

TRICHORRHEXIS NODOSA

This is an interesting condition found in the hairs of the beard and whiskers, and very rarely in those of the scalp or other parts. The hairs affected are marked at irregular intervals with whitish transverse bands, which give to them a porcupine-quill aspect. These bands or nodes under the microscope show that the cortical fibres of the shaft are frayed out in a regular manner, so that the nodal swelling resembles closely the effect produced by placing two blunt camel's-hair pencils end to end. The shaft of the hair breaks at the node when the slightest traction is made upon it, and each end of the divided node resembles still more closely the free end of the camel's-hair brush, only that it is whitish, and seems as if the hair had been recently singed.

Upon a single long hair from the beard as many as twenty nodal swellings may be counted ; this, however, is rare, as the brittleness of the shaft usually leads to the shedding of the free end, but it is common to find as many as six such markings upon the stumps of the hairs in the whiskers when these are thick and curly. They begin usually at some distance from the follicle, but may be found occasionally as near to it as one-third of an inch. In some cases the shaft may be found increased in diameter, at the point where the node is about to form, before any fraying-out of the fibres has become visible. The root-sheath is normal ; there is obvious loss of pigment at the node, in both the cortical fibres and medulla, and the latter is atrophied owing to the drying-up of the pith-cells. Otherwise the hair-structure may be in every part quite normal and apparently well nourished. Sometimes the nodes are seen in ill-formed and stunted lustreless hairs ; but many of these may in the same beard or whiskers be found free from nodosities, and the same remark applies to the longitudinal splitting sometimes observed.

Much difference of opinion exists regarding the pathology of the condition, some observers maintaining that it is of the nature of a trophoneurosis, whilst others adhere to a parasitic origin, and state that the affection is contagious.

As far back as 1879 the writer investigated this condition, and the results are embodied in a paper in the February number of the 'Dublin Journal of Medical Science' of that year. He examined carefully 300 individuals who did not shave the face, and in every one of these the characteristic nodes were found upon some hairs ; in one case only was the marking not quite typical, but the specimen was unmistakable. The most usual site is in the hair of the whisker just below the lobule of the ear, and another common place is over the region of the depressor anguli oris muscle, but he never found these hairs in the scalp or in any other place. The distribution of the affection was demonstrated by a microscopic examination of the beards of the members present at a meeting of a medical society, when perfect specimens were obtained from a dozen individuals examined. The writer quotes from the paper just mentioned : 'When I first noticed the condition, the idea of mechanical twisting being the cause seemed obvious, but I marked certain hairs close to the affected ones upon my own face and pulled at them for a few weeks without producing any effect whatever. This, with the fact of their being so rare in the hair upon the lip, satisfied me that *torsion* played no part in the production of the lesion. The universal distribution of trichorrhexis nodosa suggests that it might, strictly speaking, have no pathology ; can we call a condition, which is so general, a disease ? The explanation which appears the most likely to be correct is this: The

outer fibre-cells of the cortex of the shaft of a growing hair become hard and firm, as do those of the nails ; they do not yield to the expansion of the inner quickly growing cells of the medulla, and the result is their gradual separation and the disintegration of the cortical structure. There is unmistakable evidence of a prior enlargement, and abnormal dryness makes these cells more prone to split up ; they are more abundant in the cleanly, who freely use soap and water. The condition is not induced by the broken-down health occurring in typhoid fever and lingering diseases.'

The above extract is reprinted, as many dermatologists still speak of the condition as a somewhat rare disease in Great Britain, though more common on the Continent.

It is to be differentiated from the interesting condition first described by Professor Walter Smith and named *Monilethrix*, in which the shaft of the hair presents a series of spindle-shaped enlargements or fusiform nodes, connected together by a series of constrictions which run for the entire length of the hair. The condition is found in all the hairs of the body, and is seen soon after birth. An hereditary history is obtainable, in the majority of cases running through several generations ; in a few cases the affection has been first observed to appear later in life, after severe shock. It is regarded as of the nature of a trophoneurosis, the constricted portions of the shaft being the result of atrophic changes.

TRYPANOSOMIASIS

The condition in the human being is described under 'Sleeping Sickness,' p. 1599.

TUBERCULOSIS

Under 'Phthisis' the general etiology and pathology of tuberculosis are detailed so fully that little remains to be said under the present heading. Moreover, the other chief localised forms of chronic tuberculosis are described likewise under their own headings in the articles on 'Laryngitis,' 'Peritonitis,' 'Meningitis,' 'Lupus,' 'Compression Myelitis,' &c. The condition known as *scrofula* has no signification beyond that of a chronic tuberculosis affecting the lymphatic glands. There remains the condition to be considered which is known as :

ACUTE GENERAL MILIARY TUBERCULOSIS

This is an acute affection, characterised by the simultaneous distribution of miliary tubercles throughout most, if not all, of the organs of the body, and usually ending in death in a few weeks.

Etiology and Pathology.—The disease may occur at any period of life, but it is much more frequent in the early years, the majority of the cases, when the meningeal form is included in statistics, occurring before the fifth year. The introduction of the bacillus in food—as for example in tubercle-laden milk—may be regarded as an improbable immediate etiological factor. Such a method of infection, owing to the arrest of the micro-organisms by the mesenteric glands, produces in the first instance a localised lesion, but at a later stage the bacilli may be carried into the circulation, and the meningeal type of the disease be produced. The same remark applies to infection by the pulmonary route, since the miliary tubercles simultaneously appear in the spleen, kidney, and other organs shut out from the possibility of external infection. The infection is certainly by the route of the vessels—thoracic duct, veins, or arteries. The close relationship between tuberculosis of the arteries and veins and acute miliary tuberculosis was established by Weigert; he found that miliary tubercles were deposited both in the inner coats of the vessels and also in the adventitious layer, and that these tubercles were older than those found in the organs. The source of the bacilli he regarded as caseating extravascular foci, which, involving the adventitia as a tuberculous peri-angeitis, finally flooded the blood with the organisms which had multiplied in the vascular walls after producing tubercles in the intima.

In other cases a caseating mass ulcerates immediately into a large vessel, as into the pulmonary vein, and the bacilli in millions are distributed by the blood-stream in the aorta and its ramifications to every region in the body, without the intervention of a tuberculous peri- or end-angeitis. When the focus directly erodes the wall of any other vein near the heart, the shower of bacilli is carried in the first instance to the right auricle, and thence being pumped by the right ventricle to the lungs these organs receive the main dose; some escaping through the pulmonary capillaries are swept onwards in the blood-stream to distant regions.

Erosion of the portal vein or any of its branches may in a similar way lead to the greater number of the bacilli being arrested in the liver. Perforation of the thoracic duct will obviously convey the larger number immediately to the lung, whilst erosion of arteries, which, however, is a rare event, will be followed by a more or less localised distribution unless the perforation occur in the immediate neighbourhood of the heart.

In all cases, therefore, acute general miliary tuberculosis may be regarded as the immediate result of a previous necrosing focus of tubercle somewhere in the body, which, either by sudden perforation directly floods the blood with bacilli, or

by a slower process invades the vascular coats, and at a later date showers the contents of these secondary vascular tubercles into the blood-stream. The condition not rarely supervenes upon chronic phthisis; much more frequently it follows tubercular-gland disease in children, after the debility produced by measles, scarlatina, and pertussis. It was many times witnessed after the early heroic doses of tuberculin, and it is not rare after partial operations on tubercular bone disease and lymphatic glands. The supposed sequence of miliary tuberculosis in typhoid fever is probably explained by the fact that many of these cases are examples of tubercular disease which, in the earlier stage of the fever, were erroneously supposed to be typhoid. The bacilli have been demonstrated in the blood; but it may be safely assumed that they do not multiply in the blood itself, this liquid and the lymph merely acting as carriers of the micro-organisms to the tissues and organs affording suitable culture-grounds for their development into miliary tubercles.

The appearances after death vary according to the mode of infection and the variations produced by the methods of distribution. Thus, in a typical case where a large shower of bacilli have been abruptly introduced into the greater circulation by the perforation of a pulmonary vein, the miliary granulations are everywhere of the same age and size, resembling the minute particles of very fine sago, or, if death has been less rapid, they may be somewhat larger and with distinctly yellow centres, though uniform in size and distribution. Where the condition has been the result of successive discharges of bacilli, either from vascular tubercles or from foci in glands or other organs, several generations of tubercles may be sometimes distinguished by their sizes and by the progress which the caseating process has made in the different organs or tissues. The structure of the tubercle has already been described in the article on 'Phthisis,' and the appearance in the different organs need not be referred to. The lungs in the great majority of instances suffer most heavily, but practically every organ and tissue in the body is involved. Of those which suffer least may be mentioned the skin, muscles, ovaries, and uterus. The frequency with which the vascular choroid is involved is a diagnostic point of great importance. The changes occurring in the tubercles, as described under 'Phthisis,' obviously are not witnessed in acute miliary tuberculosis owing to the short duration of this disease; but careful exploration of the dead body usually reveals some focus or foci from which the acute invasion has sprung. These will exhibit the usual degenerative processes often in different stages, in different areas, in close proximity to each other. In the writer's remarkable case of chylous ascites, referred to in the first volume of the present

work, the primary focus was discovered in the walls of the thoracic duct.

Where the 'irruption site' is in the intima or adventitia of small vessels this focus may be easily overlooked, but an older lymphatic-gland nidus, from which the secondary vascular tubercle originally started, may be discovered.

CLINICAL TYPES OF ACUTE GENERAL MILIARY TUBERCULOSIS.—Three characteristic types of the disease are met with: (1) The *Meningeal* form, in which the cerebral symptoms overshadow all other evidence of tubercle deposition anywhere else. This is described under 'Meningitis, Tubercular,' p. 1019. (2) The *Pulmonary* form. This is more common after childhood, at which stage of life the meningeal is by far the most frequent. The condition is referred to in the description of acute phthisis, with which, however, it should not be confused, as miliary tuberculosis of the lungs may exist without the deposition of tubercles elsewhere, and the pulmonary form of acute general tuberculosis implies in its title the more or less universal distribution of the bacilli through the body, though the symptoms are those mainly caused by the lung implication. (3) The *Typhoidal* form, in which death may occur from profound toxæmia before any local evidence is established of the presence of tubercle in any organ or tissue.

Symptoms of the Typhoidal Form.—As implied in its name the symptoms are those of a severe attack of enteric fever. The onset may be even more sudden than in a serious attack of this disease, and in a typically rapid case it may set in when the individual is in apparently robust health. The clinical picture may be conceived as identical with the condition which was often observed during the reactionary fever following heroic doses of the old tuberculin. The sudden perforation of a large vein in the immediate vicinity of an old cheesy focus showers into the blood a quantity of living and dead bacilli, together with a copious dose of their toxins. The symptoms from the very start are mainly if not exclusively produced by the toxins or proteids which had been previously pent up in the old caseating focus; but as the liberated bacilli soon multiply in their separate areas in the tissues, successive large doses of toxins are absorbed, and the fever is kept up more or less continuously. Its range and the severity of the other constitutional symptoms will obviously depend upon the site and the amount of the dosage, and to a slighter degree upon the individual resistance.

Though, as stated, the onset may be abrupt, more frequently it is preceded by a period of several days in which malaise, anorexia, and muscular debility may be experienced. Fever is the most constant and characteristic symptom throughout. It may reach 103° within twenty-four hours after the onset, which is not uncommonly marked by a smart rigor or succession

of chills. The type may be continuous for several days, with evening exacerbations and morning remissions, so that the chart may present the typical features of a typhoid invasion. This, however, if watched closely, and if the thermometric observations are made at four-hourly intervals, soon reveals the true characteristic nature of tubercular fever, which is decidedly a pyrexia of *irregular* type. An evening temperature of 103° or 104° may be followed by a morning drop to 98° or lower, and sometimes the *inverted* type of pyrexia may be present for several days together, in which case the evening register may be normal, whilst the morning temperature reaches as high a range as the afternoon pyrexia of typhoid fever. Not rarely hyperpyrexia alternates with collapse-temperatures, as sometimes witnessed in exaggerated examples of hectic fever. Cornet emphasises the important fact that implication of the brain is often followed by an inhibition or depression of the fever, and frequently introduces marked fluctuations into the temperature-curve.

Whilst the range and irregularity of the pyrexia afford the most reliable means of gauging the severity and nature of the affection in the great majority of cases, it must be remembered that very occasionally an almost apyrexial temperature has been recorded throughout its fatal progress, though such cases, nevertheless, suffer intensely from prostration and tachycardia, and rapidly emaciate.

The fever is very often associated with profuse sweatings, even when there is no approach to collapse-temperatures, and sudamina, erythematous, herpetic, and other skin-eruptions are not uncommon.

The pulse is almost invariably greatly quickened, and often rapid out of all proportion to the degree of fever; it is also small and compressible, and steadily weakens as the effect of the toxins upon the cardiac muscle becomes established. When the cerebral meninges are deeply involved some slowing of the pulse-rate may, however, be observed.

The respirations are usually also markedly increased in frequency, and commonly cough is an early symptom. This is at first frequent, dry, and hacking, and only in the later stages (except when bronchitis is present) does a scanty mucoid or blood-streaked expectoration appear. No bacilli are found in the frothy sputa, unless when the general invasion supervenes upon a chronic phthisis.

Dyspnœa is a very common symptom and may amount to orthopnœa, and usually some cyanosis becomes evident about the lips, ears, and finger-tips. The breathing towards the end often exhibits the Cheyne-Stokes character. Delirium sometimes follows the headache, which is commonly present throughout, but unless when meningeal implication is marked the patient's delirium is transitory, passing off after thorough

awakening from sleep or drowsiness. The general tendency is less in the direction of active delirium than towards an apathy and stupor which gradually develop into a progressive coma, often complete for a short period before death.

The usual accompaniments of fever, as dry brown tongue, anorexia, nausea, and sometimes severe and continuous vomiting, are present. Constipation is the rule, but occasionally diarrhœa, not unlike that witnessed in typhoid fever, appears and renders the diagnosis still more puzzling. The first symptom may be a copious diarrhœa with frequent bloody stools. Albumin is frequently found in the urine, but seldom to any marked amount; when the sweating is very profuse the renal secretion may be in complete abeyance for twenty-four or more hours. The degree of emaciation before death is usually extreme, and generally, like the pulse-rate, it is out of proportion to the fever-range.

Physical Signs.—Excluding those cases where the general tuberculosis supervenes upon a chronic phthisis, there are usually no signs whatever in the thorax to warrant a diagnosis of miliary tuberculosis till the disease has progressed for some weeks. Even when cough is frequent and severe, dyspnœa marked, and cyanosis obvious, one of the most characteristic features of the disease is the negative results of the chest-examination. In bronchial cases—viz. where the deposition of miliary tubercles is associated from the onset with marked bronchial irritation—there may be râles and rhonchi from the start, but only such as might be expected in any feverish condition associated with irritation in the larger tubes, and such as might be looked for in measles and typhoid fever.

At a later stage nothing can be appreciated by inspection, palpation, or percussion, but auscultation may reveal the presence of puerile or rough vesicular breathing and fine pleural friction-sounds, soon followed by minute crepitant râles, heard over scattered areas in both sides of the chest, whilst sonorous and sibilant rhonchi at other places drown these latter sounds. Where life is prolonged for several months the signs of softening may be heard towards the end of the disease, or at any period the signs of a pneumonic consolidation may suddenly supervene. Where, however, the pulmonary manifestations set in early and the signs in the lung become marked, the case should be regarded as an example of the pulmonary, and not of the typhoidal, type of acute tuberculosis, the distinguishing feature of the typhoidal type being, as already stated, the clinical picture mainly of a tuberculin-toxæmia without obvious chest-signs. It is, however, apparent that the one type may pass into the other when life is prolonged beyond a few months, and either of these types or forms may be suddenly transformed into a

typical example of the meningeal variety of the disease by the advent of a shower of bacilli upon the cerebral tissue and membranes.

Symptoms of the Pulmonary Form.—These need not be enumerated in detail; the clinical picture resembles that just described, with the difference that the toxæmia is not so intense, and the pulmonary symptoms and signs are marked at an early stage. These latter correspond with those found in the acute forms of phthisis, and there may be evidence of extensive pneumonic consolidation, with rusty sputum. The cough, dyspnoea, and cyanosis are, in proportion to the fever and sweating, much more severe and predominant than in the typhoidal form; the disease, though an acute one, runs a less rapidly fatal and progressive course, and death often results from pulmonary œdema.

The duration of acute miliary tuberculosis will obviously depend upon many circumstances, the chief of which will be the site of the greatest deposition of the tubercles and the amount of bacilli in the focus whose introduction has set up the disease. Thus in the meningeal form death usually occurs within two or three weeks; but in some typhoidal cases, where a large amount of dead bacilli and toxins have been introduced at once through an extensive perforation, a fatal issue has been known to occur within a few days or even within a period of twenty-four hours. The severe forms of the disease commonly prove fatal within three or four weeks, and the pulmonary type may last as many months.

Diagnosis.—The positive diagnosis of acute tuberculosis is always fraught with great difficulty unless in those cases where the patient's life is prolonged to the period at which softening of the tubercles occurs. During the early stages a diagnosis can only be arrived at by a process of exclusion, unless (1) the tubercle bacilli are discovered in blood drawn from the finger or lobe of the ear, (2) or detected in a sample of cerebro-spinal fluid, or (3) when tubercles are visible in the choroid upon ophthalmic examination. It is obvious that for diagnostic purposes the use of tuberculin is entirely out of court, and the interpretation of the opsonic index is difficult under the pyrexial conditions maintaining in an acute general infection. The serum-diagnosis by means of the agglutination-reaction, when carried out under the careful precautions recommended by Arloing, may prove to be of value; in the present state of this method little reliance can be placed upon its results. The detection of the bacilli in the blood is the pathognomonic test, but unfortunately the examination usually gives negative results, because the bacilli do not remain and multiply in the circulating fluid, out of which they are strained by the capillaries of the tissues and organs after several hours, or certainly before many days.

Calmette's ophthalmo-reaction may prove useful in these cases.

As a rule, by a process of careful exclusion all diseases can, after a number of days' watching of the case, be eliminated, save one affection—viz. *typhoid fever*. In this differential diagnosis the following points are *against* typhoid: the absence of a history of exposure or the presence of a history of a previous attack, marked irregularity in the type of the fever, absence of rose-spots, of splenic enlargement, of diarrhœa, of meteorism, and of the other signs and symptoms commonly observed during the course of the disease. The fact, however, of greatest moment is that typhoid fever may exist without any sign or symptom whatever save that of the continuous fever, except the pathognomonic sign of the presence of the typhoid bacillus in the blood, urine, and fæces, and the agglutination-reaction test of Widal. If these latter tests are available and a positive result obtained, the diagnosis of typhoid fever is certain; but too much importance should not be attached to Widal's test alone. In considering the case from the tuberculosis standpoint, upon the other hand, little importance should be attached to the chest-signs and symptoms; exactly the same auscultatory evidence may be available in typhoid fever, with its accompanying bronchial catarrh, lobular pneumonia, hypostatic congestion, &c. The pulse in typhoid fever is considerably quickened, but perhaps there is no pyrexial disease in which the pulse-rate may be so frequently observed to maintain a lower level in proportion to the fever than in typhoid. This would be a more helpful point in the differential diagnosis were it not for the fact that sometimes in acute tuberculosis a meningeal invasion occurs which lowers the pulse-rate to that observed in these aberrant cases of typhoid in which it does not rise above eighty throughout. The pulse in typhoid is, however, very rarely so rapid as in tuberculosis, except after the third week, and it is always more or less markedly dicrotic during the first two weeks. Many cases of typhoid occur abruptly, with intense headache, active delirium, or other head-symptoms; and the same remarks apply also to acute tuberculosis, where the first signs may be meningeal. The pseudo-meningeal symptoms of the onset of typhoid fever lessen, however, after a few days, and often they are preceded or accompanied by epistaxis—conditions not met with in acute tuberculosis.

The great increase in the respiration-rate, orthopnœa, and cyanosis are presumptive evidence of tuberculosis only when observed in a given doubtful case in which there are no corresponding evidences in the chest *signs* to account for them. The chest *symptoms* usually do not appear in typhoid fever till after the disease has lasted some time, whilst they may

usher in the onset of tubercular fever. Splenic enlargement, when well marked at an early stage, is a sign more characteristic of typhoid than of tubercular fever.

Of great importance in summing up the evidence in favour of the case being one of acute miliary tuberculosis is the due weighing of information obtainable from the history of the patient. A marked family-history of phthisis is of some value, but a previous hæmoptysis, tubercular adenitis, or old bone-lesion is still more suggestive. The discovery of a latent apical lesion or of mesenteric-gland disease or a swollen testicle will often alter the balance. Even the history of one or more attacks of pleurisy with effusion is very significant, and not very rarely a careful examination of the spinal column reveals a slight angular curvature, caused by recent or old-standing tuberculosis of the vertebral bodies which had never been suspected. Some cases, notwithstanding the closest study and scrutiny of all the symptoms, remain incapable of being differentiated till the autopsy reveals the lesions of typhoid fever or the presence of miliary tuberculosis.

Prognosis.—The disease must be acknowledged to be always fatal. The most that can be hoped for in any case is that the acute toxæmia may lessen as the system becomes accustomed from day to day to the successive doses of the poison or as the toxins in the initial dose become eliminated by the activity of the excretory organs. Should such amelioration of the patient's condition take place there may be a prolongation of life till the newly formed tubercles soften and effect their destruction of the pulmonary or other tissues, in which case the most that can be expected is a transformation from the typhoidal to the slower but equally fatal pulmonary type of the disease.

Treatment.—This resolves itself into the judicious treatment of the fever, sweating, dyspnœa, cardiac failure, and other symptoms by such agents as experience justifies. Cold sponging, hypodermic injections of strychnine, oxygen-inhalations, and the free use of stimulants may do much to alleviate the patient's distress and prolong life for a brief period under more endurable conditions than if the victim be abandoned to his fate or drugged by agents administered in the vain hope of arresting the development of the invincible bacilli.

TYPHLITIS. (See under APPENDICITIS, p. 58.)

TYPHOID FEVER

Known also as Enteric Fever, this is an acute infectious disease characterised by the presence of continued fever, a rose-coloured exanthem, diarrhœa, tympanitis, and enlarged spleen, and accompanied by marked intestinal and other abdominal lesions caused by the invasion of the blood and tissues by the *bacillus typhosus*.

Etiology.—The disease occurs in all the habitable parts of the globe, being endemic in most temperate climates. Sporadic cases occur from time to time in cities and districts where the affection cannot be strictly regarded as endemic, and these often lead to extensive epidemics in the presence of imperfect hygiene. In war its prevalence sometimes destroys more individuals than the sword and the rifle, as proved amongst the British force in the late Boer campaign.

Age is a material factor in the predisposition to the disease, the great majority of cases occurring between the years of fifteen and thirty. Though many children between the fifth and tenth year suffer, the disease is rare in infancy, and its incidence markedly diminishes after forty-five or fifty, though occasionally it may be met with during the sixth decade. Both sexes are about equally subject to the disease, and all classes of society are liable, the poor as a rule suffering less than the rich. Season has a marked influence over its incidence; thus the disease is always more prevalent in the autumn, especially after hot dry summers; but often severe epidemics extend throughout an entire intensely cold winter, especially in cities. The disease attacks new-comers more frequently than old residents, and the writer has frequently observed that an individual may remain insusceptible to the affection when residing in a typhoid environment, but catches the infection upon his return after a long holiday. Individual and family susceptibility is often noticeable, and this is probably intensified by over-work and exposure to other depressing causes. One attack as a rule confers life-long immunity, but second attacks do very occasionally occur. There is always a doubt about the correctness of the diagnosis in seizures occurring for the third time; the writer has only met with one individual who evidently suffered from three attacks of the disease. Owing to the universal prevalence of the disease in cities, no individual can be supposed to escape all through his life exposure to the risks of the bacillus; nevertheless the majority of subjects never contract the affection. It is possible that an *acquired* immunity may account for these instances, though there is no reason to suspect that the individuals have suffered from a form of the disease so mild as to have escaped recognition. This view

is supported by the observation above-mentioned, which applies to individuals who leave a typhoid-infected district or house for a considerable period and are seized with the disease a short time after their return ; it also receives support from the fact that the bacilli have been found in the stools of healthy individuals in close contact with typhoid patients. It may also explain the fact that new-comers from healthy rural districts are so prone to become infected soon after their residence commences in a city in which the disease is endemic.

SOURCES OF INFECTION.—There being no longer any doubt about the specific nature of the *bacillus typhosus*, the manner in which the microbe is disseminated and gains admission to the human organism becomes a question of the most vital importance. It is equally certain that the chief source of infection exists in the stools passed by typhoid patients.

Before the discovery of the specific bacillus, Murchison maintained that the disease occurred *spontaneously*, the infective material arising from decomposition of the materials in sewers, soils, and animal substances being conveyed in sewer gas or emanations ; hence he named the disease Pyogenic Fever. This view is no longer held ; but in one detail of his theory his view receives support from several recent observers, who assert that after the lapse of time the virulence of the bacillus increases enormously in the fæces, being practically inert at the moment when they are discharged from the patient's body, and they become increasingly virulent after a period of twelve to twenty-four hours.

Soil-pollution affords an obvious explanation of the spread of the disease, and though the bacilli have been said to disappear from the stools after these have been kept isolated for several days at a temperature of about 60° F., when they enter the soil their presence and virulence have been demonstrated after the lapse of a year. The soil is, moreover, exposed to contamination from the urine of patients long convalescent from the disease, since numerous observers have detected the bacillus sometimes in pure culture and in enormous numbers in the urine many months after the disappearance of all fever and other symptoms. In one case investigated by Dr. Thomas Houston the bacillus was demonstrated in the urine three years at least after convalescence from a possible typhoid attack.

The condition of the soil as regards the presence in it of other sewer-contaminations—cesspools, decomposing animal and vegetable remains, &c.—is of importance, since these supply a favourable nidus for the growth of the bacilli ; but the organisms cannot, according to the results of experimental research, develop in the deeper layers of the soil. Pettenkofer, however, maintained that the bacilli develop in ground-

soils accessible to air and water; after the excreta have become virulent by the influence of the soil the bacilli are carried upwards in the air, ascending from the ground-soil, and thus convey the disease into houses. When the ground-water is high the development of the bacillus is supposed to be much retarded, as fewer cases usually occur; but in some epidemics the opposite condition has been found to exist, so that little importance need be attached to the influence of the subsoil-water. The influence of the water in the soil is, however, of much importance from another point of view, since it may be the means of conveying the bacillus into wells or other receptacles of water used for drinking purposes.

Aërial Transmission or *Air-borne Typhoid* has long been a subject of serious controversy; the majority of authorities do not consider that the disease is *contagious* in the strict sense in which this term is used. There are few cases which support the air-borne theory, and still in many hospitals the disease is treated in the general medical wards, though it is highly probable that such a method will receive universal condemnation at no distant date. The disease is undoubtedly conveyed to the healthy by direct contamination of the fingers through soiling by the excreta; even thermometers have been proved to convey the infection when used in the mouth or rectum. Innumerable instances are on record where the infection has been conveyed directly through soiled linen.

The question of whether the bacilli can be carried in the air by the emanations arising from the patient's breath or skin is another matter; and though obviously there is every possibility of their being inhaled in the fine spray ejected in the act of coughing, it seems highly improbable, except in cases of close personal contact, that this method of infection really occurs. The fermentation which arises in sewers and cesspools into which typhoid excreta have found their way may by the explosion of the numerous air or gas bubbles generated disseminate the bacilli, but obviously the limited range of such dispersion in such cases is hardly worth consideration. The commonly held view that sewer-gas cannot of itself convey the disease is probably correct, but the writer witnessed a severe epidemic unmistakably conveyed in this way amongst the nurses in an isolated fever-block. A jet of waste steam was conveyed into the main house-drain, which forced all the traps and filled the wards with a warm moist atmosphere as dense as a London fog. Nearly every ward-nurse who had not suffered previously from the disease became affected, and some patients convalescing from scarlatina also caught the infection. The entire block of two storeys remained infected for a couple of years in spite of all methods of disinfection, though a case of the disease had

never been known to arise in the building during the previous quarter of a century. This may be regarded as an instance of air-borne typhoid, but the obvious factor in the dissemination of the bacilli was the fine spray of partially condensed steam, which was able to hold the virus in suspension and lodge it in every crevice in the building. By another method there can be little doubt that the disease may be spread by the air, as witnessed in the epidemics amongst the British soldiers engaged in the Boer war. Owing to the prevalence of sand-storms the excreta deposited upon the surface of the ground and the dust, previously moistened by urine and rapidly dried by the sun's rays, were blown about and inhaled by the lungs, or, finding their way into food, became a source of infection when swallowed.

Transmission by Water, or Water-borne Typhoid.—This is undoubtedly the method by which the disease is transmitted in the majority of great epidemics, and it explains most of the sporadic and endemic examples of typhoid fever commonly met with. The bacilli find their way from the soil into wells and reservoirs supplying drinking-water, being often conveyed by streams at a distance. The contamination is usually effected by storm-water washing the excreta in the soil or upon the surface of the ground into the rivulets, and when the subsoil-water is low it drains directly into the ground-springs which go to form the water-supply. The worst possible condition of matters is where the excreta are thrown direct into the streams feeding the reservoirs. In the Maidstone epidemic of ten years ago nearly 2,000 individuals were affected by typhoid fever through the contamination of the water-supply by surface drainage.

It has been long known that under ordinary circumstances the bacillus typhosus rapidly disappears from water and sewage, and Goodall quotes Fehrs as having recently demonstrated that their disappearance is due to the presence of infusorial and other organisms in the water, the bacilli having been found within the body-wall of certain flagellata. When the water is sterile the organisms remain virulent for weeks, unless the water be exposed to the direct rays of the sun, which in summer destroys them after several hours. In this connection a very interesting case of contamination of distilled water is recorded by Pierce and Thresh. Were it not for the action of the sun's rays and the presence of protozoan organisms in the water of such rivers as the Thames which are used for drinking purposes, the ravages of the bacillus would be incalculable. Rivers fed by iced water usually suffer little effective contamination, though the bacillus will live long at a temperature below freezing-point, and the disease has been caused by eating infected ices. Probably the explanation of their attenuation under these

circumstances is the influence of their free exposure to air and sunlight.

Transmission by Food.—Milk-borne typhoid is a very common phenomenon, innumerable localised epidemics having been traced to an infected milk-supply. The old view that the disease was communicated in the milk through the cow suffering from typhoid fever is no longer held. The bacilli in milk-borne typhoid nearly always gain their admission to the milk through contaminated water used in washing the milk-cans, and very often the source of this latter is a well in the farm-yard or pasture-field. So that it may be accepted that almost all cases which can be traced to the milk-supply are really instances of water-borne typhoid. The cream of such milk is also infective, and it has been proven that the bacilli may retain their virulence for several days in butter. In many instances infected milk when made into ice creams has been known to transmit the disease. It is also quite possible that milk may be directly contaminated independently of water: the writer has seen the full milk-crocks kept for the making of butter stored beneath the bed of a farmer suffering from the disease, and the unused milk sent down from the sick-room may be easily conceived to be a source of danger; the bacilli flourish luxuriantly for a long time in milk. Next to milk-infection the most common form of food-contamination is seen in oysters, cockles, and other shell-fish collected from sewage-beds. Watercress conveys the disease more frequently than is usually realised, as this vegetable flourishes in streams contaminated by sewage, and its thorough cleansing by water is almost impossible. In the same manner lettuce, cress, celery, and all vegetables eaten uncooked may convey the disease through contamination of the soil in which they are cultivated. Strawberries and wind-blown fruit may possibly be conveyers of the disease by the same methods.

Food is readily contaminated by flies and other insects which feed upon the typhoid excreta, and this method of transmission of the disease was frequently observed in both the late Boer and Spanish-American wars. Moreover, the power of flies to infect sterile culture-media has been demonstrated, according to Osler, in the Chicago epidemic by Alice Hamilton. In the Boer war, as already stated, the dust-storms contaminated the food before, during, and after the cooking process; the flies assumed the proportions of a plague in the crowded tents, and the water-supplies were highly infected by the excreta and urine of typhoid patients, so that almost all of the recognised methods of transmission of the disease were in full operation, with the result that the deaths from typhoid amongst the British soldiers amounted to 8,000.

Typhoid-carriers. — A. and J. Ledingham have recently reported (B. M. J., January 1908) the cases of three asylum inmates to whose presence several outbreaks of typhoid since 1893 have been traced. One had been resident for eleven years and no history of an attack of the disease was obtainable, another had typhoid twelve years before, and the third suffered three years previously. Typhoid bacilli were found in great numbers in the fæces of all three, the blood reacted, and the strain from the stools agglutinated with antityphoid serum (1 in 7,000). Kayser's Strassburg case, in which every new employee in a bakery became infected, is equally remarkable; the proprietress suffered from typhoid ten years before, and numerous bacilli were still to be found in her stools. The great majority of these chronic typhoid-carriers are women, and it seems almost certain that the bacilli continue to exist and multiply in the gall-bladder. Dehler has recently successfully performed cholecystostomy in two asylum carrier cases. To the same category belongs Mackenzie and Battle's case, where infection occurred through handling the dressings from an abscess existing three years after a typhoid attack. Houston's (in 1899) is, as far as the writer knows, the first recorded case of a chronic typhoid carrier.

BACTERIOLOGY.—Since the discovery of the *bacillus typhosus* by Eberth its morphological and developmental characteristics have been worked out by a host of observers, who have established the fact that it is the specific cause of typhoid fever and is an organism of a species distinct from the *bacillus coli communis*, with which it is constantly associated and with which it has been long confounded. It is a short, thick, rod-shaped organism, one or both of whose rounded extremities are seen to contain a refractile spot, formerly regarded as a spore, but now recognised as due to a degenerative change in the protoplasm which causes it to retract towards the poles of the bacillus. In length it measures about $3\ \mu$, its short diameter being less than one-third of this, and it is readily stained by the aniline basic dyes and fails to take the stain by Gram's method.

Young cultures show the bacillus to be actively motile, and under suitable staining methods long and numerous delicate mobile flagella may be observed attached to the ends and to the sides of the rod. The bacilli when cultivated outside the body in suitable media often form threads, consisting of the rods placed end to end. The cultures perish after less than half an hour's exposure to a temperature of 140°F. (60°C.), and within a few minutes when exposed to the boiling-point of water. They flourish best at the temperature a little below that of the normal body, but may be subjected to the freezing-point for considerable periods without impairment of their vitality, though they will not

grow below 48° F. They develop best in cultures in the presence of oxygen, though growth can also be maintained in its absence; thus the bacillus is both aërobic and anaërobic. Of considerable importance is the fact that cultures perish when exposed from four to ten hours to direct sunlight, and Richmond found that in sterilised water under similar conditions the bacilli were killed in one hour. They have been found to remain alive in sterilised river-water for more than two months, but in ordinary river-water they rapidly disappear, as already stated, owing to their being eaten up by protozoa and infusoria. In sterilised stools they remain active for many months, and in potato-cultures for a period of a couple of years. In ordinary cultures they resist drying for several months, unless the bacilli be in a very thin film.

The influence of antiseptic agents upon the bacillus has been extensively tried with the view of forming a basis for therapeutic methods, but though bouillon-cultures are said to be destroyed by a half per cent. carbolic-acid solution and by .004 per cent. perchloride of mercury solution, these facts afford no justification for the hope that the bacilli can be destroyed *in situ* in the intestinal canal by any antiseptic solution which can be used with safety. Their rate of development may possibly be retarded to a trifling extent by the use of β -naphthol or calomel in full medicinal doses.

The bacillus is obtainable during life from the blood, spleen, fæces, and urine; it also exists in the bronchial secretion, sweat, and in the site of the skin-eruption. After death it has been found constantly in the Peyer's patches, mesenteric glands, spleen, and in practically every organ and tissue of the body. Experimental demonstration of the specific nature of the bacilli has been recently made by injecting and administering cultures to animals; in the case of the chimpanzee, Grünbaum produced typical typhoid fever by injecting 1 c.c. of a broth-culture twenty-four hours old. Necrotic intestinal and other lesions resembling those found in the disease in man have been sometimes observed in the lower animals after the administration or injection of the toxins, but owing to the fact that most animals do not suffer from typhoid fever (they appear to be immune to it), the absolute demonstration of the bacillus typhosus being the specific agent was not conclusive till the researches conducted by Chantemesse upon monkeys were corroborated by the previously mentioned authority. These results, combined with the absolute constancy with which the bacillus is found in the body in typhoid fever, settle the question finally. The chemical poison separated by the bacilli appears to be of the nature of a toxalbumin, but in all probability several distinct toxic substances are elaborated during the development of the microbes, and these are rendered more virulent by the presence of the bacillus coli.

From the nature of the intestinal condition it is obvious that the infection must be often multiple.

The question must, however, be considered unsettled as to whether the intestinal lesions of typhoid fever are in a strict sense primary or secondary—viz. whether the bacilli first enter the blood-stream as in a general infection and afterwards invade the intestinal glandular apparatus, or whether, reaching the intestinal glands in the first instance, they increase there and are absorbed thence into the blood-stream. Some interesting conclusions have been arrived at by Coleman and Buxton from a scrutiny of the blood-examinations made at different periods of the disease. These results, in the writer's opinion, tend to support the view that the disease is of the nature of a true general infection in the first instance. Thus the bacillus was found in the blood in 93 per cent. of the cases examined during the first week, whilst only 76 per cent. of the examinations made during the second week and 56 per cent. during the third week demonstrated the presence of the bacillus in the blood, and in one case the specific organism was found as early as the third day. Moreover, some cases have been published in which the bacilli have been detected in the blood of patients dying early in the disease, whilst no lesion of the intestinal mucosa could be detected. Nevertheless there can be no doubt that in the great majority of instances, if not in all, the poison enters the system through the alimentary tract. The few cases where pulmonary symptoms have been observed early in the disease have been adduced as proof of the possibility of the bacilli having entered by the bronchial tract.

Pathological Anatomy.—The most characteristic lesions are found in the alimentary tract at the lower part of the ileum, being especially always well marked in the neighbourhood of the ileo-cæcal valve. The large bowel suffers less frequently and less severely. The diseased process is confined to the *Peyer's patches* and the *solitary glands*, though a general catarrhal condition is usually also present, extending much beyond the limits of the affected follicular tissue. From the condition of the lesions the different stages of the process show that the changes commence near the valve at the end of the ileum and progressively advance upwards till they end somewhat abruptly about the junction of the upper two-thirds with the lower third of the small intestine.

(1) The *Hyperplastic* or stage of *Infiltration*. This is the first stage of the change; it commences with hyperæmia, followed soon by an infiltration of the solitary glands or the patches by numerous lymph-corpuscles and some larger multi-nucleated epithelioid cells, some of which may be seen to contain in their interior red blood-discs. By the invasion of the neighbouring mucous, muscular, and sub-serous layers

of the bowel by these lymph-corpuscles the blood-vessels are compressed and the initial hyperæmia diminishes, so that the swollen and prominent follicles appear somewhat anæmic. The patches stand out prominently from the level of the mucous membrane, to the extent sometimes of a fifth of an inch; the solitary glands may be enlarged to the size of small peas, and, being more deeply embedded, project usually to a lesser extent above the swollen and somewhat softened mucous membrane. In stained sections the bacilli are seen in considerable numbers in the interior of the follicles and in the lymphatic vessels. This stage commences about the fourth or fifth and reaches its complete development some time about the tenth day or a little later, when *resolution* in mild types of the disease may set in by a rapid degenerative change of a fatty or granular character, which ends in the absorption or clearing off of the hyperplasia, leaving the patch in a reticulated or pitted condition; sometimes minute extravasations and almost microscopic erosions occurring. In the majority of cases, however, the second stage sets in about the tenth day. This is known as the stage of

(2) *Necrosis or Sloughing*. The newly formed lymph-cells perish by coagulation-necrosis induced by the bacilli and their toxins, and the result is the production of a greyish-yellow slough, which may correspond to an entire Peyer's patch with its superimposed mucosa. The solitary glands at this stage are the seat of a similar necrotic process, each follicle becoming covered by a small yellowish-green coating. The larger sloughs slowly separate, the necrosed tissue being loosened first at the margins; an entire patch may be thus shed in one piece or in fragments. Sometimes during the previous stage of hyperplasia two or more Peyer's patches may become involved in one continuous mass of lymph-cell infiltration, and the whole piece may necrose, forming one extensive slough.

The depth of the necrosing area varies greatly, in some cases apparently only involving the superficial stratum of the mucous layer, whilst in others the slough consists of the necrosed muscular coat and the tissues down to the peritoneal coating of the bowel. Before this stage is completed the sloughs have usually become dark or almost black through staining by bile and blood, and the lower end of the ileum may become the seat of a large black eschar as if charred by heat.

About the middle of the third week the sloughing stage is completed by the entire separation of the necrosed portions, but it is not unusual to see different stages of the process side by side within an area of a few inches. As a rule, the shedding of the sloughs formed in the solitary glands occurs later than in the patches, and these generally necrose only

within about a foot from the ileo-cæcal valve and are more liable to be affected severely in children.

(3) *Ulceration Stage*.—By the third week the typical typhoid ulcer is met with; its shape and size depend upon the site of the necrosis in the lymphoid tissue. Where this has been in a solitary gland the ulcer is circular, whilst the necrosis of a Peyer's patch eventuates in an elliptical or oval ulcer situated at the part of the bowel most distant from its mesenteric attachment and with its long diameter corresponding to the long axis of the tube. When the necrosis has been confined to a portion of the patch a small circular or irregular-shaped ulcer results, and it is very common for a number of such to be seen separated by areas of unabraded mucous membrane over the site of a patch.

The size of the ulcers varies; where an entire patch has sloughed or several fused patches have been involved a large surface of several inches in length may be denuded. A Peyer's patch in the healthy bowel is of oblong form and, according to Symington, measures from half an inch to two or even four inches in length and from half an inch to about an inch in width, and is placed lengthways in the intestine, opposite to and distant from the mesentery; whilst the solitary glands are scattered over every part of the mucosa as well at the mesenteric as at the free border, and both between and upon the valvulæ conniventes. This distribution explains the varieties in shape, outline, and magnitude of the ulcers found in typhoid fever. The ulceration is always most advanced and most extensive, both in the solitary glands and in the patches, within the last foot of the termination of the ileum, and this in rare and severe cases may be involved in one continuous sheet of ulceration; more frequently the ulcerated surface, however, in such cases is broken up by bridles or irregularly shaped areas or islands of mucous membrane. In the majority of mild cases, however, the ulcers are comparatively few and of small dimensions, and decrease in size as they are traced upwards in the bowel.

Their edges are softened, never hard or infiltrated, and they are usually more or less undermined, so that their swollen purple fringes float when the portion of opened and excised bowel is placed in water. The base or floor of the ulcer may be the mucosa, the muscular layer, or the peritoneal coat. In a large ulcer these different depths to which the necrotic process has extended may be witnessed in different portions of the denuded area, whilst if the isolated ulcers be examined their depths may often be observed to decrease as they are traced from the neighbourhood of the valve upwards. The floor of the ulcer is usually smooth, never markedly indurated or dense, and is upon the whole most frequently formed of the exposed muscular layer of the bowel.

(4) *Stage of Cicatrisation.*—This begins soon after the shedding of the sloughs in the middle of the third week, and is complete usually within ten days or a fortnight, unless the necrosis has been both deep and extensive. The sore becomes gradually covered by a layer of granulation-tissue arising from the margins of the ulcer and growing towards the centre, the mucosa finally covering it with an epithelial layer as the granulation-tissue develops. No puckering or contraction of the lumen of the gut results from the cicatrix, which is smooth, somewhat depressed in the centre, and slightly pigmented. Unless the necrotic process has been deep and wide the normal constituents of the mucosa and submucosa appear to be reproduced, so that usually no traces of the former ulceration are visible if the bowel be examined after death arising from some other cause within several months of the convalescence from the fever, though evidences of deep ulceration have been known to remain for years after the attack, when this has been prolonged and severe.

In about 30 per cent. of the *post-mortems* made in typhoid fever similar changes to those above described are found in the upper part of the large intestine where the solitary glands necrose and circular or oval ulcers form with their long diameter stretching across the lumen of the ascending colon. Very rarely is the colon ulcerated when the small intestine is but slightly involved; cases have been very occasionally met with in which the Peyer's patches in the small intestine and the solitary glands in both the large and small bowel have appeared normal or but slightly hyperæmic, when the mesenteric glands have shown unmistakable evidence of the disease and the specific bacillus has been found in the blood. The vermiform appendix is rarely involved, but sometimes it is seen to perforate in severe cases. Where death has occurred from perforation in the late stages of typhoid fever the usual changes in the peritoneum which accompany perforative peritonitis will be evident.

Mesenteric Glands.—These pass through a hyperæmic and hyperplastic stage similar to that observed in the intestinal follicles during the first ten or twelve days of the fever. The changes are always best marked in those glands whose distribution corresponds with the lower portion of the ileum and the upper part of the colon, but the retroperitoneal glands are generally affected also. The glandular swelling usually begins to resolve by a process of fatty degeneration before the intestinal lesions are healed, but septic infection from the ulcers may lead to softening and suppuration, and the glandular structure may necrose and pass through stages similar to those observed in the patches and solitary follicles. Unless rupture of a necrotic area and peritoneal inflammation ensue, the large softened glandular tumours become harder,

and finally remain for a long time firm and indurated ; occasionally the exudation or pus passes into the stage of calcification, lime-salts being deposited in the inspissated contents.

Splenic Changes.—These are as constant as the intestinal lesions ; the organ swells at a very early stage of the disease so frequently as to constitute a valuable diagnostic sign. The enlargement is never excessive ; the organ softens and may be found of the consistence of a soft pulp in some cases, the leucocytic infiltration being always excessive. Infarcts are common, so that rupture may occur ; though the writer only once witnessed death from splenic rupture in typhoid fever. The bacilli are always found in great numbers in the swollen organ, even in the earliest stage of the disease.

The *bone-marrow* and the *lymphoid tissue of the tonsils* show changes similar to those observed in the solitary glands, patches, and spleen.

The *liver* is often, but not always, obviously swollen ; though somewhat hyperæmic in the early stages, it is usually paler than normal at the autopsy. Nodular areas of necrosis or of lymphoid tissue occur amongst the liver-cells, which usually show marked parenchymatous degenerative changes. Hepatic abscess and acute yellow atrophy have been occasionally observed as complications. Cholecystitis is more common, and this not rarely ends in suppuration, produced either by the bacilli or by the virulence of the bacillus coli, which often shows itself in the typhoid-infection. The catarrhal condition of the bile-ducts sometimes causes jaundice, and the retention of the secretion ultimately tends towards the formation of calculi.

Renal Changes are of importance, since albuminuria is frequently observed during life, but sometimes the *post-mortem* characters of the kidneys afford no explanation of this. The organs are enlarged, more or less congested, and the epithelial lining of the convoluted tubes shows the signs of granular degeneration and cloudy swelling in most instances. Sometimes the typical changes seen in acute Bright's disease are found. The bacilli are usually present in large numbers, which may account for the condition in which numerous small cell-accumulations have been observed clustered round the finer blood-vessels, and which occasionally eventuate in miliary abscesses in cases of multiple infection. The bladder is often found in a state of catarrhal inflammation ; occasionally a diphtheritic deposit has been found upon its lining membrane and upon the lining of the renal pelvis.

Heart.—The heart shows always more or less change ; in many protracted cases it is obviously degenerated, being flabby, pale, or yellowish-brown in colour, and distinctly softened or quite friable. Under the microscope fatty or granular parenchymatous and, much more rarely, amyloid

degeneration may be seen in such cases ; but in some instances of ordinary duration, where marked myocardial weakness has been a prominent symptom during life, the microscope may reveal no evidence of structural change in the fibres. Sidney Martin has isolated a toxic substance from the bacilli which produces marked fatty degeneration of the heart when injected into animals; hence it is probable that at a late stage of the disease, even in those who recover, more or less structural change in the myocardium may be safely assumed.

The endocardium is affected in a considerable proportion of cases, and a condition apparently identical with that observed in malignant endocarditis is sometimes found. The bacilli have been detected both in the myocardium and in the vegetations deposited upon the inflamed valves. Pericarditis probably only occurs in multiple infections.

Arterial Changes are even more constant than the cardiac, signs of recent endarteritis being present in the vessels, especially of the lower extremities, and the bacilli are constantly found in the proliferations on the inner coat as on the endocardium. The lumen of the vessels may be obstructed by thrombi laden with the bacilli, as in ulcerative endocarditis; but in this latter affection the blocking is arterial, whilst thrombosis of the veins much more commonly is witnessed in typhoid fever than arterial obstruction. The most serious phase of the endarteritis is met with when the coronary arteries are involved. The condition of the blood will be referred to later on.

Pulmonary Changes are common; lobar pneumonia is often met with of pneumococcal origin, but more frequently the lesions characteristic of disseminated bronchopneumonia are observed. Hypostatic congestion and splenisation, and bronchial changes are very common; occasionally pulmonary infarcts, abscesses, and gangrene of the lung-tissue are present. Empyema, pleurisy, and enlargement of the bronchial glands are also sometimes found. Not infrequently *laryngeal ulceration* and perichondritis, with or without necrosis of the cartilages, are observed; and thrombosis of the laryngeal vessels in the mucosa, or deeper, is stated by Chevalier Jackson to be the most frequent local initial lesion. A diphtheritic exudation upon the laryngeal mucous membrane is not uncommon.

The *Muscular System* shows the changes found in all protracted pyrexial affections, more or less granular degeneration being evident in the voluntary muscles, and the bacilli have been found in the muscular substance, sometimes in association with hæmorrhages or abscesses.

The *Nervous System* exhibits no specific changes beyond those sometimes induced by the thrombotic process occasion-

ally attacking the cerebral arteries and venous sinuses. Meningitis is rarely present, though the symptoms during life often arouse a suspicion of its presence ; the bacilli have, however, been found in the exudate, which in rare instances has been reported as purulent. This latter event is probably the result of a mixed infection, though the capacity of the specific bacillus for producing pus-formation is undoubted.

Symptoms.—The *incubation* stage of typhoid fever is a period of variable length. In the majority of instances it may be taken as practically the same as that usually observed in small-pox—*i.e.* thirteen clear days. It has been found to be as short as five and as long as twenty-eight days. As in the other exanthemata, this period is devoid of symptoms, though, owing to the difficulty in fixing the advent of the next stage, the end of the incubation-period is said to sometimes be accompanied by sensations of languor, malaise, &c. These really belong to the next stage.

The *invasion* is most indefinite and insidious in the majority of instances, so that it is the rule with many physicians when investigating the history of a doubtful case of fever to regard a typhoid diagnosis as improbable when the patient can give a clear history of having taken ill at any definite hour in a particular day. There is, however, a sufficient percentage of cases in which the invasion is better marked—a fact which should necessitate caution in the routine acceptance of this rule.

Headache, general muscular pains, anorexia and nausea, chilly sensations and sleeplessness are commonly experienced for two or three days before the patient takes to his bed or seeks advice. The date of the insidious invasion is usually calculated from the time the patient takes to bed, whilst some authorities in their description of the stages of the disease count the invasion as dating from the first headache or chilly sensation so that considerable discrepancies creep into statistics which largely deal with questions of duration and the advent of particular symptoms and signs upon certain days.

An initial rigor of a distinct type, with cold, hot, and sweating stages, is seldom witnessed at the invasion ; the chills are subjective. The headache is sometimes severe, and this is very striking in a small group of cases which begin abruptly with cerebral symptoms. The initial headache lasts usually but for a few days, and is always frontal. There may be diarrhœa or constipation, with or without abdominal pain, but a slight degree of tympanitis associated with some tenderness in the right iliac fossa is more common. The tongue is furred, but always moist at this early stage and clean at the tip and edges ; the breath is often offensive ; and the combination of these signs with a dull expression of countenance is sometimes characteristic. A sign of considerable diagnostic

value is smart *epistaxis*; according to some observers this occurs in nearly one-third of all cases. The pulse shows acceleration early and is as a rule dicrotic; but a very significant fact of diagnostic importance sometimes from the onset is that, though the pulse is quickened, it often is not accelerated in proportion to the degree of pyrexia present. Ker therefore emphasises the importance of these signs of early typhoid in the statement that 'a pulse which presents this want of relation to the temperature and at the same time is dicrotic should also suggest that the fever is enteric.'

The *temperature* is found elevated before or as soon as the patient takes to bed, and the chart of a typical case, when viewed throughout, presents usually very characteristic features. The rise is steady and continuous for the first four or five days, the evening temperature exceeding that of each corresponding morning by about $1\frac{1}{2}^{\circ}$ or 2° F., and upon each morning there is usually a drop of about 1° from the previous evening pyrexia, so that by the fourth or fifth day the temperature of the evening reaches in a typical smart case to 103.5° or 104° . At this figure it remains more or less stationary, with slighter morning remissions, for a variable number of days, usually about seven. This stationary period, called the *fastigium*, is followed generally by a period of three or four days in which the type of the fever becomes more remittent, the morning temperature showing a fall of a couple of degrees F. The middle of the third week is thus reached, after which till the twenty-first day the intermittent type is approached, in which the morning temperature gradually approaches the normal, though the evening rise still registers 2° or more higher.

This range varies very considerably in different cases, and sometimes, instead of the above-described gradual *defervescence* which terminates in the normal evening temperature being reached by about the twenty-first day, there may be before or after this period a crisis accompanied by a sudden fall to the normal or below it; this is, however, very rarely as marked as the corresponding crisis observed in typhus. Complications seriously disturb the characteristic temperature-curves; thus sudden hæmorrhages or perforation may rapidly cause a drop to sub-normal, and the advent of pneumonia sometimes raises the temperature to a hyperpyrexial range. In some epidemics the duration of the fever in apparently uncomplicated cases extends into the fourth or fifth week or even longer, and in mild cases the duration of the pyrexia may be indefinitely prolonged by septic complications or sequelæ. In rare cases the disease has been known to pursue an almost apyrexial course throughout, and in one fatal case in a medical man under the writer's observation the temperature only rose above the normal just before death from meningeal

trouble, though the intestinal ulceration was found in an advanced stage at the autopsy. Other points regarding the temperature will be mentioned later on.

From the above review of the course of the temperature throughout the disease the consideration of the reader may be turned to the condition of the patient soon after he has taken to bed in a typical attack of typhoid fever.

During the first week the anorexia remains well marked, but thirst is present, though nausea may exist; the tongue gradually tends to become dry and the lips are parched. The skin is hot and usually dry, though sweatings more or less profuse may occur from time to time. Diarrhoea may develop before the end of the week, but this symptom is very variable; there may be two or three loose motions in the twenty-four hours, or constipation may prevail. The patient does not appear usually to be seriously ill, and often expresses himself as being only out of sorts even when the early frontal headache may remain, though lessened in degree; the pulse is quick and the temperature high, and there may be slight wandering at night; cough is not unusual and the epistaxis may recur.

By the end of the first week or the beginning of the second, if the abdomen be carefully examined the following signs may be obvious. There is some tympanitic distension, with tenderness in the right iliac fossa, in which region careful palpation may reveal gurgling, and the spleen will be found enlarged and palpable below the left costal arch. A sign known as that of Bernard may be present at this stage, which disappears within a few days later; it consists in the presence of two or three small almond-shaped swellings, lying parallel to the long axis of the colon in the region of the right iliac fossa, and separated from each other by a distance of half an inch or one inch. They are supposed by some observers to be the hyperplastic Peyer's patches in the lower part of the ileum.

The Eruption.—The date of the first appearance of this varies considerably, but it may be accepted as a general rule that its usual appearance is from about the seventh to the ninth day or a little later. Very rarely is it observed before the fifth day, and in such instances it may be assumed that the disease is of longer duration than the history of the case seemed to warrant. Occasionally, however, its advent is delayed till the middle of the third week. It is often absent, especially in children; and in the writer's experience the very frequent absence of the eruption has characterised several epidemics of the disease within recent years.

The front of the abdomen and chest are the most common sites, but it is very often first detected upon the back, possibly being rendered more obvious by the pressure caused by the

dorsal decubitus. It may be sometimes seen upon the thighs, but very rarely upon the arms, hands, or face. The characters of the eruption when typically marked are pathognomonic. It consists of a series of small round or oval rose-coloured papules, which continue to come out in successive crops. Only a few—half a dozen—may be present at any one time, but sometimes as many as a hundred have been counted. Each spot measures about from 2 to 4 mm.—*i.e.* from .08 to .15 of an inch, the majority being about a line in diameter. It is distinctly appreciated as being raised above the level of the skin when the finger-tip is lightly passed over it, and it does not feel as hard as a lichen or acne papule. A more important distinction, however, is that the typhoid papule invariably disappears completely upon pressure, from its first appearance till finally vanishing into a faint scarcely appreciable stain. When the finger-pressure is removed the rose-colour begins to return almost immediately. The average duration of each papule is about three or four days, and usually they may be seen fading as new ones begin to appear. The crops continue to appear all through the second and third weeks, and a fresh crop may be often observed to come out after the fever and other symptoms have completely disappeared. In rare cases the rose-papule becomes capped by a minute vesicle, but petechiæ never occur in the spots, even when, as in very rare conditions, petechiæ have been observed in other parts of the skin. A sign known as Phillipowicz's has recently been observed as occurring about this period: it consists in a well-marked yellow discoloration of the skin on the palms and soles, and is best marked in the case of children. This sign is claimed by Regis to be pathognomonic; it lasts throughout the attack, disappearing after the fever and showing itself again in any subsequent relapses.

During the progress of the second week all the symptoms, save usually the headache, become better marked. The pulse quickens to 100 or 110, but becomes less microtous as the third week is approached. The diarrhœa, when present, assumes more or less typical characters, the motions being more numerous and thinner in consistence and yellow in colour, somewhat resembling pea-soup. They are usually highly offensive in odour, contain the bacillus in great numbers, are alkaline in reaction, and sloughs may be detected along with numerous small flakes of lymph, mucus, and often small blood-clots.

The fever by the middle of the second week shows the characters already mentioned in the description of the typical chart. It is at about its maximum height in the evenings, and the morning remissions may be somewhat better marked, though in severe cases they are slighter in range. The respirations are quickened with the pulse-rate, but not very markedly

so ; cough and some mucous expectoration are commonly present. Mild delirium may appear at night and during the daytime ; the facial expression of the first week changes to one of marked dulness and apathy, a condition intensified by the deafness often present. The tongue begins to get dry and brown ; the cheeks are pale, with often a hectic flush on the malar bones. The abdominal distension, notwithstanding frequent copious motions, increases, the spleen becomes more palpable and ileo-cæcal gurgling more marked, though this sign should never be elicited at this stage on account of the danger of perforation.

Before the advent of deafness subjective sensations of tinnitus may be severe and disturbing. The urine has the well-marked characters of the pyretic condition : it is high-coloured and scanty, often contains a trace of albumin, and the diazo-reaction of Ehrlich is usually present.

In severe cases hæmorrhage from the bowel may occur about the end of the second week. In mild examples of the disease the symptoms begin to clear up and the temperature may fall upon the fourteenth evening, but these fortunate occurrences are decidedly exceptional.

During the third week the abdominal symptoms in severe cases deepen, the diarrhœa being often profuse, in some cases only making its first appearance at this stage ; and hæmorrhages from the bowel are common. The tongue is now either dry and brown or beefy-red and sometimes fissured, sordes accumulate about the teeth, gums, and lips, there is obvious general wasting, and commonly copious perspirations occur, often causing profuse sudamina. The pulse has become still more rapid, showing marked feebleness and loss of diastole ; the heart-sounds are weak. The temperature keeps high in the evenings, but in the average of smart cases the morning remissions are well marked, as the evening temperature gradually falls towards the normal before the commencement of the fourth week. Delirium, which was usually mild and transient during the second week, now becomes more or less constant ; it is usually of the low muttering type and often associated with subsultus or muscular tremors, and alternates with somnolency or occasional bursts of active delirious excitement. The dangers of perforation, pulmonary complications, collapse from cardiac failure or profuse intestinal hæmorrhage reach their height towards the end of the week.

The fourth week in the majority of cases is ushered in with a gradual improvement in all the symptoms ; the pulse slowly falls in frequency and increases in volume as the evening temperature becomes normal. The tongue cleans, and a moist condition of the mouth is noticeable as appetite returns. The urine becomes more abundant and pale, and ceases to deposit urates on cooling, and the bacilli are abundant

in it. The cerebral disturbances gradually fade away as natural calm sleep returns. Bed-sores may, however, make their appearance at this stage, if not noticed during the third week. The abdominal symptoms slowly disappear, often constipation setting in, sometimes suddenly, without the motions having shown any gradual tendency to increase in consistence.

In severe cases the condition of the patient during the fourth week is an accentuation of that observed during the third, the cerebral symptoms assuming the character of a more profound toxæmia, as seen in muscular tremors, somnolence, unconscious evacuation and retention of urine. Symptoms of pulmonary and cardiac embarrassment show themselves by lividity, great frequency and feebleness in the pulse and breathing, the patient finally passing into the so-called *typhoid state* and succumbing in a comatose condition apparently more from the profound influence of the toxins than from any direct result of the prolonged high fever. This result is, however, not seldom witnessed during the third week, and may even occur as early as the end of the second, or may be deferred till the fifth or sixth week, especially in cases showing relapses.

The convalescence is always slow and protracted; for months and sometimes for a few years afterwards the bacilli may be found in great numbers in the urine or in the contents of periosteal or other abscesses which may remain as sequelæ.

VARIATIONS IN THE CHARACTER OF THE ONSET, in the type and in the course of the fever are more common in typhoid than in any other pyrexial disease.

The Onset.—(1) This, as already incidentally mentioned, may be suddenly ushered in by intense agonising headache, sometimes accompanied by vomiting, photophobia, active delirious mania, or retraction of the head, muscular twitchings, and convulsions, simulating meningitis or spotted fever. This form of onset is usually referred to as the *cerebral* or *cephalic*.

(2) A *pneumonic* type is seen occasionally in which the first symptoms of onset are apparently identical with those observed in acute lobar pneumonia or pleurisy. Pneumonia in typhoid, however, is rarely due to an initial pneumonia setting in along with the true enteric seizure; when lobar pneumonia occurs in the disease it is in the great majority of instances a late event, but Dreschfeld regarded some of these cases as due to the early invasion of the pulmonary tissue by the *bacillus typhosus*. (Pneumonia is, however, much more frequently mistaken for typhoid fever than the latter disease is mistaken for pneumonia.) This form of onset is frequently described under the name of *Pneumotyphoid*.

(3) A *bronchial* type of onset is occasionally observed in which the invasion-symptoms are those of a severe epidemic catarrh, with more or less pharyngeal redness and tonsillar swelling, or the latter signs may be alone present or are only accompanied by the slight initial catarrhal symptoms so often observed in the typical insidious onset of the disease.

(4) A *gastric* type of onset is more common, the first symptoms being those of intense and frequently repeated vomiting, sometimes associated with considerable abdominal pain and diarrhœa. In severe epidemics caused by serious contamination of the water-supply such cases have been at first regarded as due to poisoning by some mineral irritant.

(5) A rarer form of onset is that which begins with renal symptoms closely simulating, if not identical with, acute Bright's disease, the urine being highly albuminous, smoky or bloody, and containing numerous tube-casts and epithelial detritus. This condition is sometimes styled *nephro-typhoid*, and may be regarded as practically identical with the renal complication which is not a very uncommon event during the end of the second week, but which is seldom accompanied by bloody urine. It is probably caused by the sudden violent irritation produced by the bacilli or their concentrated toxins before the renal cells have become accustomed to their action, as they apparently do in the later stages of the disease and after convalescence.

(6) Other rare forms of onset are those ushered in by *convulsions*—an event sometimes seen in children; by *laryngeal symptoms*, and by marked *rigors* resembling the onset of malaria. The onset by signs of *epistaxis* is so commonly observed as occurring in the typical insidious invasion of the disease that as a type of onset it should not be mentioned, but very occasionally severe continuous or frequently recurring epistaxis may be the only symptom of the onset observable for several days.

(7) The *latent* form of onset is by no means uncommon, though it is usually described as a type of the disease and not as a variation in the mode of the invasion. It is known as *Ambulatory typhoid*. The patient does not feel really ill, but keeps upon his feet, often pursuing his usual avocation till some serious complication sets in, as intestinal perforation, or a formidable hæmorrhage occurs. The mortality is usually very high in these cases with latent onset, and the dangers increase with the length of the latency.

VARIATIONS IN THE TYPE AND COURSE OF THE FEVER have been incidentally mentioned, and the cases, as in every other fever, are classified as mild and severe. In the mild type the temperature does not exceed 103° throughout, and in the evenings may have fallen to normal about, or even before, the end of the second week; but the severity of the fever

often bears no relation to its duration: smart or even severe pyrexia may terminate satisfactorily within fourteen days, whilst a low range of fever-temperature may be protracted into the sixth week or longer. It does not assist the physician as regards prognosis to classify typhoid fever into *mild*, *typical* or *ordinary*, and *severe*, since a fatal perforation occurs sometimes in cases of such a mild type that the individual has followed his occupation till the rupture. Nevertheless for convenience it is necessary to recognise the form or type of enteric fever usually described as the *severe* or *grave* variety, but it is advisable to limit the term to those cases which show more or less gravity from their beginning. The severity which intensifies the clinical picture is usually manifested in the high range of the fever, but not necessarily so, as severe toxic symptoms may appear early and remain throughout with a moderate temperature.

A subdivision of the grave form is recognised under the title *Bilious typhoid*, in which the gastric symptoms are usually marked from the onset by repeated vomiting, which soon is associated with the vomit becoming bile-stained or consisting almost entirely of apparently pure bile, as in sea-sickness. The headache is usually very persistent in this serious variety, and cardiac feebleness becomes well marked.

Ataxic typhoid or *Cerebro-spinal typhoid* is the name given to those cases in which the cerebral symptoms—delirium, mania, convulsions, &c.—appear early and run into the typhoid state. The *Hæmorrhagic* form is characterised by an unusually well-marked tendency not only to bowel-hæmorrhage but to purpuric extravasations or skin-petechiæ and internal bleedings.

An *Adynamic* form is met with in which the predominant symptoms appear to be produced by the virulence of the toxins upon the cardiac muscle and nerve-centres, causing profound prostration, with early signs of heart-failure and a low type of muttering delirium soon passing into stupor. These latter forms are often classified together under the title of *Malignant typhoid fever*.

Some authorities classify the type of the disease from the peculiarities observed in its mode of onset as *nephro-*, *laryngo-*, *pneumo-*, *pleuro-*, and *pharyngo-*typhoid, but often in these cases the disease begins to run its typical course after the lapse of the first few days. Dreschfeld recognises a *spleno-typhoid* form in which there is excessive splenic enlargement and the fever is of the relapsing type. *Typho-malarial fever* is the name given to the disease when it occurs in a patient who is or has been recently suffering from malaria.

The *Apyrexial* type has been referred to already upon page 1746, and the *Ambulatory* variety has been included amongst the variations in the mode of onset.

An *Abortive* form is sometimes met with, in which often, after a smarter invasion than usual, the fever suddenly disappears, sometimes as early as the fourth or fifth day, and the case is regarded usually as one of mistaken diagnosis; but at the end of a week or later a relapse occurs and the disease runs through a typical course with an unmistakable eruption, fever, and abdominal symptoms. Though the relapse is generally very severe, these cases very rarely terminate fatally.

The term *Relapsing typhoid* has been sometimes applied to the cases in which relapses follow a distinct typhoid attack after the patient has entered into the convalescent stage. The relapsing tendency is a distinct feature of the typical disease, and may occur in any of its varieties.

Typhoid fever in children often runs the typical course observed in adults; the eruption is, however, more liable to be scanty or absent, and epistaxis and diarrhœa are less frequently marked, hæmorrhage and perforation being rare events, though the fever is usually high and the nervous symptoms more pronounced. The type of the fever is sometimes distinctly remittent—a fact which has given the name of ‘infantile remittent fever’ to many of the examples of enteric fever in infancy. By others the typhoid of childhood has been often styled *gastric fever*. Aphasia, cancrum oris, and periosteal abscesses have been more frequently observed as late complications or sequelæ than in the case of adults.

Senile typhoid often runs a more or less latent course in aged patients, since the fever is seldom high, though prolonged. The abdominal symptoms are seldom so marked as in young adults, being overshadowed by the graver symptoms due to cardiac asthenia and often a low type of pneumonia.

Fœtal Typhoid.—Hicks and French have recently reviewed thirty cases in which typhoid fever occurred during pregnancy, and in about half of these the bacillus typhosus has been demonstrated as having been transmitted to the infants, the transmission being more frequent in proportion to the lateness of the delivery. The result of the agglutination-test is interesting. In one of these cases the blood-serum of the fœtus was found to react to Widal’s test, though no bacilli were present in the fœtal blood. This would go to prove that the agglutinin may pass through the placenta; and Osler states that it may pass through the lacteal gland to the milk and a transient reaction may thus be obtained in the nursing’s serum without the infant being infected.

In the typhoid fever of pregnancy, though the risk to the mother is apparently little, if at all, increased, the majority of the patients abort, and the fœtus is very liable to perish.

Post-typhoid Fever.—Excluding cases in which genuine relapses occur after apparent convalescence has set in, Delafield

has drawn attention to the long-continued fever which is the result of sepsis following the ordinary attack of four weeks' duration. This form of septicæmia has been styled post-typhoid fever, and may exist alone or occur in the fourth week in combination with the phenomena produced by the typhoid toxins. The writer has been impressed by the frequency with which tubercular manifestations become apparent after these long-continued fevers, and believes that many of the examples are instances of a more or less general tuberculosis which originates in some latent tubercular focus.

Para-typhoid Fever is the name given to a form of continuous fever only to be distinguished from the typical typhoid by the absence of the pathological changes found in the solitary follicles, Peyer's patches, and mesenteric glands, though during life a rose-coloured papular eruption, diarrhœa, enlargement of the spleen, and most, if not all, of the main symptoms of enteric fever, are observable. These cases, however, do not respond to Widal's test. Various bacilli have been found in the blood and excretions which have been described under different names. It is still a question awaiting demonstration how far the group of organisms intermediate between *B. typhosus* and *B. coli* should be considered as specific or as developmental variations of the typhoid or of the colon bacillus. To this group belongs the *bacillus enteriditis* or the organism known as *Gärtner's bacillus*, which was demonstrated by Durham and others to be the cause of several epidemics of para-typhoid fever traced to infected meat.

RELAPSES.—At any stage of the convalescence, but most usually by about the middle of the second week, after the evening temperature has become normal, the typical relapse may set in and the patient may pass through what may be described as a second attack of typhoid fever, with all the characteristics of the temperature-curve, enlargement of spleen, rose-coloured eruption, diarrhœa, &c. As a rule, this second attack or relapse runs a shorter and milder course, but there may be a second or even a third relapse. In the typical abortive form of typhoid already referred to, the first attack is short and the relapse is severe and prolonged, but, like the relapses occurring in all varieties of the disease, they are very seldom fatal. The occurrence of relapses is more common in some epidemics than in others; hence their frequency varies much in statistics—from 3 or 4 per cent. to 12 or 15. The higher figures probably include cases of spurious relapses—viz. those in which from some indiscretion in diet or too early getting out of bed there is a slight irregular rise of temperature for a few days; thus it is a common occurrence for a mild degree of fever to follow the

return to solid animal food when this has not been gradual, but such recrudescence should not be regarded as of the nature of a true relapse, since the temperature speedily returns to the normal upon a return to liquid diet. In rare cases a relapse may set in after a month's absence of all fever and abdominal symptoms, even after the convalescent patient has resumed his ordinary vocation. There is no reason to suspect a renewal of the infection in these cases, though the cause of the second attack or relapse is difficult to explain; probably it is due to some temporary breakdown in the conservative process of immunisation.

THE BLOOD-CHANGES IN TYPHOID FEVER.—Reference has already been made to the fact that the bacilli are found in the blood even at a very early stage of the disease; in the great majority of instances they can be demonstrated during the first week, and often as soon as the patient takes to bed. They are much less frequently found during the fourth week, but they return to the blood during a relapse.

As a rule there is no leucocytosis present in the uncomplicated disease; the total number of white cells is in fact always reduced—a point of very considerable diagnostic importance. Moreover, the differential count gives fairly definite results even at an early stage of the fever. In normal blood the polynuclear neutrophiles average about 70 per cent. and the large mononuclear and transitional cells not more than 2 to 4 per cent. In typhoid fever the latter are often increased to over 20 per cent., and the polynuclear neutrophiles fall to under 60 per cent. Higley also found that the eosinophiles were reduced from their normal 2 to 4 per cent. to 1·7 per cent. In the leucocytosis which occurs in septic and inflammatory conditions the polynuclear cells are always markedly increased and may number eight to ten times that of the other cells; in the same manner, when a septic complication arises in typhoid fever the number of these cells with multipartite nuclei may become greatly increased.

The red blood-discs are in normal number in a given quantity of blood during the first and second week, but in the third there is usually marked anæmia, the number sinking to 3,500,000 or even less in the c.mm., and the fall in hæmoglobin is always relatively much greater.

Widal's Reaction or the Agglutination-test is based upon the influence which the serum of a typhoid-fever patient's blood possesses over the specific bacilli of the disease. Thus when the bacilli taken from a young vigorous culture are added to the diluted serum of typhoid blood they rapidly lose their motility and undergo agglutination, as evidenced by their aggregation into clusters or clumps. The test is carried out in the laboratory, the physician taking a sample

of the patient's blood at the bedside by puncturing the fingertip or ear-lobe and permitting the blood to flow into a bulbous glass pipette. The serum, having separated from the coagulum, is diluted (1 in 30 to 1 in 60), and to this is added a drop from a young bouillon-culture or an agar-emulsion of the bacillus typhosus, and the mixture upon a microscopic slide is placed under a cover-glass and examined by a power of 250 or upwards. Within about half an hour, when a serum-dilution of 1 in 30 has been used, a positive reaction shows the bacilli congregated into clumps, and motionless. A control-experiment in which serum from a healthy subject is used instead of the serum from the suspected typhoid case will show the bacilli actively motile for many hours or even days, and they show no tendency to clump or gather in clusters. With a 1 in 10 dilution clumping may be seen within fifteen minutes, and with a 1 in 50 the agglutination should be complete within about an hour.

A modified method is to use dead cultures, by which fresh cultures or the use of a laboratory may be dispensed with. The characteristic reaction can also be obtained macroscopically, but it is much slower. The process consists in mixing the diluted serum with the bacterial emulsion in a small test or U-tube, when at the end of twenty-four hours the bacilli will be precipitated to the bottom of the tube, forming a sediment visible to the naked eye.

The value of the reaction as a test for typhoid fever is doubtless very great; but though a positive result is obtainable in over 95 per cent. of cases, there are two or three fallacies which considerably impair its usefulness. Thus it is entirely absent throughout the disease in a small percentage of typical cases in which there can be no doubt about the diagnosis. It is usually well marked before the eighth day, but it may be absent at this period, though it becomes very evident during the third week or during a relapse later on. Very occasionally a positive reaction is obtainable in other diseases, but a more serious fallacy arises from the fact that it may be obtainable by using the serum of a patient who has suffered from a forgotten attack of the disease many years before.

COMPLICATIONS.—*Perforation* of the bowel is the most serious of all the numerous complications of the disease and accounts for more than 25 per cent. of the deaths caused by the typhoid bacillus. It occurs in between 2 and 3 per cent. of all cases, and, as already stated, it may be the first symptom in the ambulatory type of the disease; but the event is extremely rare before the middle of the second week, the usual period being about the middle of the third or during the fourth week. In a small number of cases perforation has occurred during convalescence, and such cases would probably be more numerous but for the precautions usually under-

taken regarding dietary. The first symptom usually is a sudden severe pain in the right hypogastric or right iliac region, which may be continuous or paroxysmal; more frequently the pain is continuous with severe paroxysmal exacerbations, and it soon tends to become generally diffused over the entire abdomen. With the first abrupt onset of the pain there is marked tenderness to pressure, but this symptom should never be elicited; soon the abdominal muscles, especially over the painful area, become board-like in their rigidity, even when the surface is but delicately palpated. Soon, almost immediately in some cases, the clinical picture becomes one of grave shock or collapse. The temperature falls, the pulse is thread-like or absent from the wrist, the features are pallid and pinched, the forehead bathed in cold perspiration, and the respirations are quickened, shallow, and thoracic. Vomiting may occur early, but often the patient seems unable to vomit, and either retching or hiccough sets in later.

These symptoms are obviously those of the acute peritonitis which always supervenes, and which soon causes marked visible and palpable changes in the abdomen. As a rule, there is seen a very considerable degree of distension, and Osler points out the importance of the increasing pain on pressure, the increasing muscular spasm, and the tension of the abdominal wall; the respiratory movements, which may at first have been restricted upon the affected side, become uniformly diminished. Gentle percussion will reveal a tympanitic note which the trained ear may often differentiate from gastric and intestinal resonance; this is obtainable over the area in which normal hepatic dullness is usually present, and there may be considerable flatness or dullness over the flanks when much effusion has taken place.

When the perforation has been of the pin-hole character the above signs are considerably masked, though the pulse is small, frequent, and thready, and the temperature falls. If the extravasation of the intestinal contents becomes shut off by rapidly forming adhesions the general abdominal tympany and diminution of the respiratory movements may escape detection, though usually some obliteration of the liver-dullness can be elicited. It should, however, be remembered that even with a perforation of considerable extent the abdomen sometimes remains flat or even retracted, with its walls in a state of permanent rigidity, and pain may be entirely absent. The urine is often suppressed. The mental condition remains clear, and sometimes pain and discomfort disappear before the patient finally sinks from profound collapse. In some cases, especially where the perforation has been small, there is distinct evidence of a reaction taking place from the initial collapse, the change being observable in a rise of temperature due to the localised peritonitis, and the pulse may

become more distinctly palpable. In a very small percentage of cases recovery may ensue.

Peritonitis.—This serious complication, though usually the result of intestinal perforation, as just described, may arise from the bursting of an abscess in a mesenteric gland, appendicular or splenic abscess, or from rupture of the latter organ, or of a suppurating gall-bladder, or much more rarely from an hepatic abscess.

General peritonitis very rarely occurs from an extension of the inflammation from the serous coat of the bowel when this constitutes the base of a deep intestinal ulcer which has not perforated; but a localised peritonitis is a very common event in such cases, circumscribed adhesions forming between neighbouring coils of bowel or between the latter and the abdominal wall. Localised pain and tenderness to pressure are usually well-marked when the parietal peritoneum is involved; sometimes a distinct friction-fremitus may be obtained before the adhesion has been completed, and occasionally this sign may be also detected some hours after a perforation.

Both in perforative and in general peritonitis occurring independently of perforation, a marked leucocytosis is usually present.

Hæmorrhage.—This, like diarrhœa, though usually regarded as a complication of typhoid, should, when limited in amount, be strictly looked upon as a clinical feature of the disease, since it occurs to an obvious extent in nearly 5 per cent. of all cases and probably in at least 10 per cent. of the grave cases in adults; it is comparatively rare, however, in children. The most common period for the first appearance of marked hæmorrhage from the bowel is at the end of the second or beginning of the third week, though often it does not occur till some time during the fourth week. When small in amount and not recurring frequently, it gives rise to no clinical symptoms, and does not appear to markedly increase the mortality of the disease; but when the hæmorrhages are frequent, or profuse though few in number, the condition must always be regarded as serious or grave.

In these latter cases the bleeding comes on as a rule suddenly, often with no preliminary warning of slight blood-staining in the stools, and sometimes even in the absence of diarrhœa and other marked abdominal symptom. The first indication may be a great fall in the temperature if this chances to be taken just after the blood has been poured out into the bowel at the routine hour of using the thermometer. The drop may amount to 5° F. or more. It is soon accompanied by a sensation of faintness or even collapse, and in rare instances death from syncope has occurred before the blood had been expelled from the bowel. The pallor of the face, the characters

of the pulse, respiration, and symptoms of collapse correspond to those observed in other instances of hæmorrhage, and are as a rule in proportion to the amount of blood leaving the vascular system, and especially in proportion to the rapidity or suddenness of the escape. The motions in the severer forms of intestinal hæmorrhage may consist of pure blood, apparently unmixed with intestinal secretion; it may be of a bright red, but usually it is dark or almost black. The active bowel-hæmorrhage may be followed for a few days by tarry motions, consisting of the blood which has undergone changes in the bowel after the cessation of all oozing or hæmorrhage; this event is more likely to occur when diarrhœa is absent.

The rare early intestinal hæmorrhages are very seldom profuse, and are usually accompanied by considerable mucus; they are the result of the catarrh or hyperæmia which is often associated with the first week of the bowel-trouble. In some cases the hæmorrhages of the early period are due to a distinct hæmorrhagic tendency, and are then found in association with skin-petechiæ, severe epistaxis, hæmaturia, or hæmatemesis, and the later intestinal bleedings may be fatally accentuated by the presence of this tendency, which sometimes manifests itself as a family trait in the disease.

Diarrhœa and Meteorism.—These can only be regarded as complications of the disease when they occur in grave degree, appearing early and dominating the clinical picture. Both these conditions tend to favour hæmorrhage and perforation: the former by hastening unduly the separation of the sloughs, and the latter by increasing the pressure of the gases within the intestinal tube. Moreover, the great distension, by elevating the diaphragm, may seriously embarrass the action of the heart and increase the respiratory trouble in the presence of pneumonia or hypostatic congestion.

Cardiac Complications.—As already stated, the dirotism of the pulse is an early feature, which disappears in the later stages. The continuous high fever tends to induce serious myocardial degeneration in prolonged cases. The first sign of this condition is witnessed in the feebleness of the first sound of the heart; later on the sounds are difficult of differentiation, the condition known as embryocardia becoming established as the diastolic pulse shortens and the contractions become accelerated. Various alterations in rhythm may be observed: bradycardia may appear as a sequel to the attack; but in some rare cases, as already mentioned, the pulse may not count more than fifty throughout the progress of the fever. With the establishment of the myocardial change, gallop-rhythm, with marked irregularity of the pulse, may appear, and signs of dilatation of the left ventricle, as seen in the supervention of a mitral systolic murmur caused

by relative insufficiency ; general lividity, with rapid breathing, soon shows itself in association with symptoms produced by the engorgement of the internal viscera.

Pericarditis is much rarer than myocardial complication, and is seldom met with, unless in association with pleuritic or pulmonary inflammation, especially in the case of children ; but in adults endocarditis of the malignant or ulcerative type, though rare, is more common than pericardial mischief, and may manifest its presence by arterial plugging and dry gangrene. These latter signs may, however, be the result of an arteritis, which is either of the obliterative or parietal type, involving the cerebral or limb arteries.

Thrombosis and embolism are more commonly due to phlebitis, the most frequently involved vessel being the left femoral vein, the clotting in which may extend into the pelvic veins. The detachment of the clot in the femoral or other vein occasionally causes sudden death from cardiac embolism or blocking of the pulmonary artery. The phlebitis is ushered in usually by a smart rigor, marked increase of the pyrexia, profuse sweating, quickened pulse, and with local pain and swelling of the extremity below the obstruction.

Pulmonary Complications.—The most serious of these is the advent of acute pneumonia. As remarked already, in a very small percentage of cases the invasion of typhoid fever sets in suddenly, with symptoms and signs of acute lobar pneumonia (pneumo-typhoid) ; this onset is due either to a possible coincident multiple infection, or to the bacillus typhosus early attacking the pulmonary tissue—more probably the latter, since the specific bacilli of the disease have been demonstrated in the sputum. The usual period of the advent of acute pneumonia as a complication in typhoid is from about the middle of the second till the end of the third week. The initial symptoms of the lung-complication are then usually masked by the primary fever and by the bronchial catarrh often present from the commencement. There may be no smart pain in the side, or rusty sputum, to direct attention to the thorax, but an increase in the pyrexia and acceleration in the respirations will be always present along with the characteristic signs of consolidation. The pathogeny of this complication is almost invariably the result of an invasion by the pneumococcus, though, as might be expected, the bacillus typhosus may also be found in the consolidated lobe, and not infrequently streptococci ; during influenzal epidemics the bacillus of the latter disease may be also found.

When these mixed infections cause pneumonia, the type is especially liable to be lobular, and sometimes pulmonary gangrene or numerous small abscesses supervene.

Hypostatic congestion is more commonly found after death than recognised during life, unless routine examination of the chest is carried out daily in protracted cases. Collapse of the lung and infarcts are also not very uncommon. Pleuritis, like pneumonia, may occur in very rare cases at the onset of invasion, but the supervention of pleurisy, like that of the pneumonia, usually occurs about the beginning of the third week; it may end in empyema, especially in the typhoid fever occurring during an epidemic of influenza.

Bronchitis is common in a minor form in most cases at the onset, and occasionally appears as a serious complication during the second or third week, when it may assume the capillary type in the very young or old.

Laryngeal Complications.—The larynx, as seen by the recent statistics of Chevalier Jackson, is more commonly invaded than is usually recognised. There is the ordinary catarrh, which may be regarded as a part of the bronchial irritation, but in his series of typhoid cases, in which the mirror was frequently used, ulceration was detected in nearly 20 per cent. The infection in this complication, as in the pneumonic, is usually a mixed one, pus cocci being frequently detected. Perichondritis also sometimes occurs, which may lead to necrosis of the laryngeal cartilages and ultimate grave stenosis. Œdema of the glottis is a very serious immediate result of the laryngeal ulceration, and may occur with or without the formation of a diphtheritic membrane. Paralysis of the vocal cords from neuritis affecting the nerve-supply of the laryngeal muscles sometimes occurs at a later stage, or during convalescence. The presence of grave laryngeal ulceration may be entirely masked till the sudden supervention of stridor, hæmoptysis, dyspnœa, dysphagia, severe pain, tenderness, or swelling over the larynx, with aphonia or croupy cough, directs attention to the condition of the throat. The perforation of a small laryngeal ulcer has been known to produce general emphysema of the cellular tissue, as pneumothorax has been observed occasionally to follow the rupture of a small necrotic area near the surface of the lung.

Liver and Spleen Complications.—The most common evidence of hepatic trouble is jaundice, usually the result of a duodenal catarrh extending up the ducts, or it may be the sequel to an attack of acute inflammation terminating in a suppurative cholecystitis; more rarely hepatic abscesses form. The jaundice may be due to the severity of the toxæmia leading to inspissation of the bile and obstruction of the minute bile-ducts. It is now a recognised fact that gall-stones are often a remote sequela of typhoid fever, the bacilli having been found in their interior. The splenic complications consist of infarctions and abscesses, and sometimes the enlarged and softened organ ruptures, causing a fatal peritonitis.

Renal Complications.—Apart from the rare mode of onset of the fever with symptoms of acute nephritis (nephro-typhoid or nephro-typhus, as it is sometimes called), the urinary system is often the seat of complications occurring at a later stage.

Retention of urine is one of the most common of the complications of grave typhoid fever, in which condition it is very liable to be overlooked, and by the inexperienced nurse and physician is apt to be mistaken for incontinence, especially when the patient is delirious or unconscious. Moreover, the delirium and stupor are intensified always by retention of urine. The bladder becomes enormously distended in some cases, and the paralytic dribbling overflow of urine is too often regarded as evidence that there can be no accumulation of secretion in the bladder. This mistake is the more likely to occur when there is great tympanitis which masks the outline of the distended organ, and the writer once saw the apex of the bladder nearer to the xiphoid than to the umbilicus in a patient who had been unconscious for more than a week. The catheter removed considerably over a gallon of urine from this patient, who finally recovered, but for many years he continued to suffer from bladder-irritability.

Suppression of urine is so rare in typhoid that retention should always be suspected if no urine be passed for twelve hours; and there is no other condition in the disease in which a daily routine examination is more imperatively demanded. Polyuria is common, especially when the patient is taking great quantities of milk and other fluids, and Osler relates one case in which forty pints of urine were passed in a single day. Pyuria is not rare; the pus is probably formed independently of the typhoid bacilli, which may appear in enormous numbers in the urine without causing any signs or symptoms of irritability of the urinary tract. The source of the pus may be either vesical or renal, more probably the former in catheter-cases; pyelitis sometimes ending in pyelonephritis, and more rarely in perinephritic abscess, occurs occasionally during the third week, and even during convalescence. It may be either unilateral or double, and is probably the result of a mixed infection, or it may be caused by the virulence of the bacillus coli, which is sometimes found in the urine in company with the typhoid bacillus. Albuminuria of a trifling degree is an almost constant feature during the height of the fever, and possesses no clinical importance, but an acute tubal nephritis, though uncommon, may set in at any stage of the disease: all authorities agree that chronic Bright's disease is one of the rarest sequelæ; possibly its presence after convalescence may be a coincidence. When the catarrhal inflammation of the bladder extends to the urethral tract, orchitis may follow, which, as in the orchitis

of parotitis, may lead to permanent wasting of the organ ; but suppuration is rare.

Parotitis, moreover, also occurs as a complication of typhoid, and is very prone to lead to serious suppuration and sloughing of the cellular tissue of the neck.

Acute mastitis ending in suppuration, *ovaritis*, and *salpingitis* are observed as rare complications.

Pyæmic Complications.—Allied to the above-mentioned parotitis, which, though usually due to extension of local septic inflammation along Steno's duct, is sometimes of the nature of a true metastatic abscess, other evidences of general pyæmia are sometimes present. The most common of these is the series of abscesses which may appear in different parts of the body and in different tissues, as in the muscles, joints, and subcutaneous cellular tissue. This pyæmic condition may originate in septic thrombi, the result of phlebitis or of arteritis or of malignant endocarditis, but it may arise from bed-sores, furunculosis, or from mesenteric-gland suppuration, or any other lesion produced by a concurrent infection with staphylococci or other pus-forming organisms.

Septicæmic Complications.—These have been already referred to in the consideration of the variations in the course and duration of the fever ; they arise during the later stage or during convalescence, and constitute the condition recognised as Post-typhoid fever, in which the only indication of the presence of sepsis may be the undue prolongation of the pyrexia, complicated by frequently recurring rigors and profuse sweatings.

Bone and Joint Complications.—The most common of these is the periostitis, which occurs sometimes in the last week or during convalescence. Any bone in the body may be involved, but the periostitis generally attacks the bones of the lower extremity, especially the tibiæ ; the ribs, however, not infrequently suffer, the ultimate result being often chronic suppuration or caries ; rarely acute necrosis may supervene. These complications are more accurately described as sequelæ, since they tend to run a very slow course and recur sometimes long after the primary attack has passed away. The bacillus typhosus has been frequently found in the lesions years after convalescence from the fever.

The joint-complications vary considerably. Some of them (the suppurative forms) obviously belong to the pyæmic category ; others appear to be of rheumatic nature ; whilst a true typhoid arthritis is occasionally met with in which a tendency to spontaneous luxation has been observed. The arthritis may be solitary or multiple ; usually only one joint is involved, and this is often the hip, in which the head of the bone is prone to slip out of its cavity. Probably of the same nature is the interesting sequela to which considerable

attention has been directed of late years by Gibney and by C. J. Wilson. This is the condition recognised as the *Typhoid Spine*, the symptoms of which first, as a rule, appear during convalescence in young male adults, usually about the period when the patient is permitted to leave his bed. After some slight exertion or movement, weakness in the back-muscles is complained of, associated with a dull aching pain, which soon becomes most acute or agonising in intensity and paroxysmal in nature, the attacks of pain being readily induced by the slightest movement, jar, or pressure, as in an examination of the spinal column. The back is always held rigid by the patient in order to avoid movement of any kind. Often there is no fever or other symptom, but sometimes pyrexia of an irregular type is present, and occasionally lateral or angular deformity may be observed, or swelling of the lumbar muscles.

In some such cases spinal-cord symptoms are present, the sphincters being weakened; anæsthesia may be detected, and the deep reflex is variously altered in the lower extremities. With these symptoms there may be a highly general neurotic condition developed, which, according to some authorities, is accepted as evidence of the affection being of the nature of a pure neurosis; whilst others regard the pathogeny of the sequela as due to periosteal involvement or to a fibrositis of the ligaments of the spinal column.

The duration of the affection varies from a few weeks to twelve months, Wilson giving the average as four and a-half months; and the prognosis is as a rule favourable when prolonged rest and fixation of the spine is effected.

Eye, Ear, and Mouth Complications.—The eye-troubles are rare, and for the most part consist of conjunctival and corneal ulceration, or paresis of the ocular muscles from neuritis occurring in the late stage of convalescence, or from weakness of accommodation setting in after protracted attacks of fever. Double optic neuritis, suppuration of the globe, and cataract have been rarely observed. The ear not infrequently suffers, otitis media resulting in cases of mixed infection. The complications which occur in the mouth and pharynx are probably always caused by staphylococci and streptococci, and resemble those met with in the larynx. A false membranous exudation is not uncommon, and this may extend downwards. More frequently ulceration of the soft palate is observed; the ulcers are multiple and small, often with punched-out margins, and they may extend into the œsophagus and larynx. It is in these septic conditions of the mouth that parotitis is likely to occur, and acute glossitis is not a rare complication. In ill-fed children *cancrum oris* of a grave type may occur during the third week and lead to extensive gangrene, destroying the cheek,

gum-tissue, and even ending in necrosis of the jaw and shedding of the teeth. Acute fatal pneumonic changes, or pyæmia, are liable to supervene in these cases, and ulceration or gangrene of the genitals may occur.

Nervous Complications.—These, though mentioned last, often figure prominently throughout the graver forms of the disease; most of them, however, can hardly be regarded as complications, since they are but an intensification of some of the symptoms usually seen in a typical severe attack of the fever.

Delirium is an almost constant symptom in all cases where the pyrexia is high and long-continued. In the form of onset with intense cephalalgia, delirium may appear upon the first day and may closely simulate meningitis. As a rule, however, it does not become marked in typical cases of severe typhoid fever till about the middle or end of the second week, or later, when it presents the usual quiet or muttering type already described. In alcoholic cases, a fierce delirium may set in suddenly, independent of pneumonia, but much more frequently the mental disturbance assumes the typical form of delirium tremens, which, in mild cases of typhoid fever occurring in alcoholic subjects, is liable to overshadow the clinical picture of the fever.

Delirium may, however, occur in a comparatively mild case independent of the effect of a general toxæmia or of pyrexia, in which case it will probably be due to a genuine meningitis caused by the bacillus typhosus or by a multiple infection. It may be the only symptom in rarer cases of cerebral sinus thrombosis, abscess, or general encephalitis. It is not rarely due to neglected retention of urine, and may pass off after the emptying of a greatly distended bladder.

Convulsions are rare. They may usher in the grave cerebral complications just mentioned, or they may be the direct result of an unusually severe toxæmia, especially in neurotic children, in whom they are more likely to occur at the onset. In a very small percentage of cases a true uræmic convulsion may be the first symptom of acute renal complication, in which case it is more liable to occur at the onset in the nephrotyphoid form.

Retraction of the head, spasms or rigidity of the neck-muscles may be a marked feature at the onset, due, according to Osler, to localisation of the typhoid bacillus in the cerebro-spinal fluid without suppurative meningitis. These cases are liable to be mistaken for epidemic cerebro-spinal fever, but lumbar puncture settles the diagnosis. In other cases tremors affecting the extremities and facial and tongue-muscles are seen; as a rule they may be accepted as a measure of the intensity of the general toxæmia, and are seen in their worst form in association with coma-vigil, with its subsultus and

carphologia. Increased irritability of the muscles is seen in all severe cases in the emaciation-stage of the third week and later, and may be elicited by tapping the pectoralis major, when a small tumour will appear, which lasts for a quarter to half a minute or longer. Occasionally the muscles may become very painful, and the seat of distressing cramp.

Hemiplegia is a rare complication. When it does occur, it usually appears late in the attack, and is liable to be accompanied by transient aphasic symptoms. It is probably caused either by septic emboli from the endocardium, or a thrombosis originating in cerebral endarteritis. Acute ascending paralysis of the Landry type has been observed, and occasionally poliomyelitis of a less extensive distribution may arise; but these events are more likely to occur as sequelæ than as complications during the pyrexial stage.

Neuritis affecting a single nerve-trunk, or two or more large cords of a plexus, is not a rare complication during the end of the second or in the third week. It is ushered in with intense pain, tenderness, numbness, and sometimes erythematous swelling of the limb, and may cause temporary paralysis of the extensor muscles, as in lead-poisoning.

Multiple neuritis is much more common, and it usually occurs during late convalescence; it may involve both the lower and upper extremities, and be followed by a considerable degree of wasting. Though the condition is usually transient, in one case the writer has known it to persist in the legs and feet for several years, seriously interfering with locomotion, and he believes it to be more frequent after typhoid fever in the tropics. The condition known as 'tender toes,' in which there is a marked hyperæsthesia of the tips and pads of the toes, sometimes involving the sole, but rarely the dorsum of the foot, is probably of the nature of a neuritis, though it usually disappears within a couple of weeks.

Rolleston has recently drawn attention to the frequent absence of the abdominal reflex in typhoid fever, as a diagnostic sign and as a guide to the condition of the patient, the return of the reflex often being an early sign of approaching convalescence.

As already stated, ocular paralysis and disc-atrophy have been observed as the result of neuritis, but the cranial nerves are very seldom affected. Disseminated sclerosis has been frequently found as a sequela of typhoid; and in many cases, especially in women, hysterical manifestations, though rare during the attack, not uncommonly show themselves in an intractable form during late convalescence. Tetany has been observed several times, and is said to be more common after some epidemics than after others.

Of more importance are the serious states of mental impairment which not rarely follow an attack of the disease. Any

of these may occur as an apparent continuation of the delirium or mental disturbances which characterised the pyrexial period, the otherwise convalescent patient never recovering his mental balance. The writer has several times witnessed a similar transition from delirium to permanent insanity in delirium tremens. More usually, however, symptoms of mental failure begin first to show themselves during or after apparent convalescence, and these may gradually deepen into mania, melancholia, or total dementia in some cases, though as a rule the outlook is better than in the psychoses occurring under other conditions.

Skin-Complications.—The most important of these are bed-sores and local gangrene, which result from pressure, especially in the cases in which profound toxæmia is marked. For the most part they are preventable, and are seldom witnessed when the nursing is skilfully and conscientiously carried out. Occasionally gangrenous patches appear upon the lobes of the ear, tip of the nose, and female genitals, and also on the cheeks.

Boils, erysipelas, herpes, urticaria, and erythematous or measly and scarlatiniform rashes are not infrequent, and erythema nodosum is often seen; but it must not be forgotten that many of the so-called skin-complications of typhoid fever are but instances of drug dermatitis, since the cutaneous system in typhoid fever easily responds to external or internal irritants, as seen in the constant presence of the *tache cérébrale*, the frequent occurrence of *maculæ cæruleæ*, *petiomata*, *taches bleuâtres*, or the blue spots which are produced by the irritation of pediculi. Petechiæ occur in the hæmorrhagic cases, and copious sudamina, or miliaria, are present when sweating is excessive.

The hair is usually shed after the attack, but permanent baldness is very seldom observed to follow. The nails are ridged transversely, as after all serious interference with the general nutritive process. An important point is the condition of the skin during convalescence, as there is sometimes a complete general desquamation, and this in children, where the duration of the attack has been short and where a complicating rash with throat-symptoms had been present at the onset, may lead to the suspicion of scarlatina, but the desquamation following typhoid is almost always of the branny or furfuraceous type, though in very rare cases extensive peeling of the skin of the fingers has been noted in children. An interesting sequela is often observed in the skin of the abdomen and inner aspect of the thighs; this consists of the presence of white lines like those which follow parturition, and which are known as *lineæ atrophicæ*: in typhoid fever they have been supposed to be the result of neuritis. Œdema of the skin may be observed from vaso-motor disturbance, a

complicating renal disease, and anæmia, or as a result of plugging of the veins in the leg, or as a result of severe mononeuritis.

Complications arising from the presence of some concurrent diseases have been already incidentally mentioned—viz. diphtheria, pneumonia, cancrum oris, nephritis, and erysipelas. Malaria occasionally complicates typhoid fever in tropical regions, as in temperate regions the disease may sometimes be associated with measles, scarlatina, and chicken-pox; but, as pointed out by Osler, there is no such thing as the hybrid disease styled typho-malarial fever.

The most important association is *tuberculosis* in any of its varieties; this may have been present in a latent form before the typhoid onset, and the influence of the toxæmia produced by the poisons manifested by the bacillus typhosus appears to rouse the quiescent bacilli of tubercle. In this connection a serious possibility has been generally overlooked which, in the writer's opinion, explains many of the difficult problems associated with the tuberculosis or phthisis so often found following typhoid fever. Owing to the enormous quantities of uncooked milk consumed during an attack of the fever and to the state of the intestine, with its numerous ulcerated areas, and to the general debilitated state of the patient, all the conditions which favour a real tubercular infection through milk contaminated by the tubercle bacillus are present. This is probably the explanation of some of the cases met with in which the symptoms of typhoid seem almost imperceptibly to glide into those of a miliary tuberculosis or acute pulmonary phthisis, so that the physician is often left in doubt whether there had been an initial typhoid invasion at all in the case.

Apart, however, from the possibility of the secondary infection being conveyed during the dietetic treatment of typhoid fever, there is always also the obvious possibility of tuberculosis developing during the prolonged stage of debility existing during the tardy convalescent period; but in this case the late advent of the tubercular lesions and their circumscribed extent are suggestive of a post-typhoid origin.

Diagnosis.—In a typical case the characteristic temperature-chart, slow onset, associated with epistaxis and followed by diarrhœa, abdominal tenderness, and distension, the appearance of the raised rose-coloured spots coming out in successive crops, and the enlargement of the spleen, leave no doubt about the diagnosis when the case has been watched for some days. In the early stage, however, the diagnosis is often beset with greater difficulties than exist in the case of almost any other febrile malady, since many of the above-mentioned symptoms, and in rare instances all of them, may be entirely absent; and without the aid of bacterio-

logical methods even at a later stage the disease can sometimes only be diagnosed by a careful process of exclusion.

As regards the temperature-curve, the dictum of Wunderlich is a very valuable guide in most instances at the beginning of the attack: if the thermometer registers 104° upon the second day typhoid fever is negatived, and the same rule holds good if the temperature does not approach 104° upon the fourth evening; whilst if, in the absence of any other discoverable cause of pyrexia, the evening temperature in a patient of middle age registers between 103° and 105° by the end of the first week, with morning remissions of 1.4° to 1.7° F., the case may be diagnosed as typhoid.

Widal's agglutination-test is of great value, but often, though found occasionally as early as the third or fourth day, a positive reaction is not obtainable till the seventh or eighth. For the methods of employing it and the fallacies attendant upon its application to diagnosis, see page 1755.

Ehrlich's Diazo-reaction.—Though found present in acute tuberculosis, typhus, measles, malaria, pneumonia, and some other conditions of high pyrexia, this test is of considerable value in the diagnosis of typhoid fever. Ker points out that its negative is greater than its positive value, since it is so frequently present between the sixth and twelfth days of typhoid; consequently a case of continued fever in which the reaction is not obtainable at this period is in all probability not one of typhoid fever. The test is applicable only to the urine, and the reaction depends upon the formation of diazo-sulpho-benzol when sulphanilic acid (amido-sulpho-benzol) is acted upon by nitrous acid. The aromatic substances present in the urine of the typhoid-fever patient combine with the diazo-sulpho-benzol to form aniline colours. For the application of the test a fresh saturated solution of sulphanilic acid in a 1 in 20 solution of hydrochloric acid and a separate 1 in 200 fresh solution of nitrite of sodium are required. A little urine in a test-tube is mixed with an equal bulk of the first-mentioned solution, a few drops of the nitrite solution are added, and the tube shaken till a froth forms upon the surface of the liquid, which is then to be made alkaline by ammonia, when both the foam and the fluid will assume a port-wine or rose-red colour if the reaction be positive, a yellowish-brown coloration without redness giving a negative result.

Bacteriological methods for the identification of the bacillus typhosus in the blood, spleen, urine, and stools, and in the rose-spots, have been extensively applied, and afford conclusive evidence in all cases when the investigation is carried out by experts in the laboratory, often a positive result being obtainable before Widal's reaction can be successfully demonstrated.

A *differential blood-count* as a diagnostic agent is more easily applied, and its value is discussed upon page 1755. The new method of making sub-cultures from the blood after great dilution by bouillon is a marked advance in the method of diagnosis in the early stage of the disease, as described upon page 1773.

The disease most likely to be mistaken for typhoid fever with severe onset is *typhus*. In this disease there is a sudden invasion, often with a prolonged rigor; the cerebral or mental symptoms soon become marked, and the temperature is high and continuous. The appearance of the typhus-rash, with its mulberry or dusky-red maculæ and its almost characteristic sub-cuticular mottling, usually settles the diagnosis upon the evening of the fourth or by the middle of the fifth day. Typhus is very rare except in epidemic form; the spots remain out till convalescence or death, and being petechial they do not disappear on pressure.

Meningitis is closely simulated by those cases of typhoid which begin with sudden severe onset characterised by intense cephalalgia. The tubercular variety of meningitis, being the most common, affords the chief difficulty. In this affection the onset is very seldom sudden, being usually preceded by a variable period of malaise in which vomiting is very often a marked feature; the temperature is seldom high, epistaxis does not occur, the pulse is often full and bounding and never markedly dicrotic, constipation is usually not only present but obstinate in degree, the abdomen is markedly retracted, and the spleen cannot be felt below the costal arch. The eye-symptoms are characteristic, photophobia, ptosis, ocular paralyses, inequality of the pupils, tubercle in the choroid, and double optic neuritis finally showing themselves.

Cerebro-spinal meningitis, in both its sporadic and epidemic forms, may be simulated by those cases of typhoid with cerebral symptoms, accompanied by marked retraction of the neck-muscles and twitchings. The differential diagnosis may only be possible after a few days' waiting; but the early appearance of herpetic and hæmorrhagic eruptions, with a marked degree of leucocytosis, the discovery of the diplococcus of Weichselbaum in the fluid from lumbar puncture, and the presence of Kernig's sign soon establish the diagnosis of cerebro-spinal meningitis.

Malaria is excluded usually without difficulty by a careful search for the plasmodium in the blood.

Influenza occurring in its gastro-enteric form, which is very common in some epidemics, is differentiated by the grave depression and prostration which are nearly always absent in the early stage of typhoid, the suddenly high and irregular type of the short-lived fever, the markedly thick

and white porcelain-like fur upon the tongue, the prominence of the vomiting, and the appearance of diarrhœa early or coincident with the onset; anorexia is usually complete, and often jaundice is present. The spleen is, however, often found enlarged at an early stage of this variety of influenza, but epistaxis does not occur, and the bacillus may be found in the sputum which is usually present.

Tubercular peritonitis is differentiated by the history of the case. Rarely does the disease appear without some clue being obtainable upon careful examination of the abdomen, a palpable tumour being usually present, consisting of enlarged mesenteric glands, adherent coils of bowel, localised peritoneal effusion, or masses of rolled-up omentum. In some cases, however, tubercular peritonitis sets in suddenly with profuse diarrhœa, or even bloody stools, high fever, abdominal pain, tenderness, and distension, so that a diagnosis is impossible without waiting for bacteriological investigation. The curve of the temperature in cases even of the most severe and rapid type will invariably be soon found to show marked deviation from the characteristic chart of typhoid.

Acute Miliary Tuberculosis.—The differential diagnosis between this grave malady and typhoid fever constitutes one of the most difficult problems in clinical medicine. The points are discussed in detail under 'Tuberculosis,' immediately preceding the present article (see pages 1730 and 1731), and need not be repeated here. The inherent difficulty of the problem lies in the fact that the two diseases are much more frequently combined than is usually realised, and, as already suggested upon page 1768, an attack of acute miliary tuberculosis may follow one of typhoid fever as the direct result of a general tubercular infection caused by the ingestion of the large amounts of uncooked milk which usually form the sole dietary during the several weeks of illness and convalescence. The new method of blood-examination affords the only means of identifying the bacillus typhosus in all ambiguous cases.

Acute Pneumonia.—Three problems in diagnosis may present themselves in this connection. A case of true croupous pneumonia may begin with an insidious onset, in which cerebral symptoms manifest themselves without the characteristic catch in the side or any physical signs in the thorax, if the pneumonia is apical or if the consolidation of the lung starts in the centre of the lower lobe. The rule laid down by Wunderlich regarding the temperature-limit may prove useful in eliminating typhoid under these circumstances, and leucocytosis will be almost certainly present. It is well to remember that the diazo-reaction may be obtained in true croupous pneumonia, and that the spleen may be also enlarged; but in a few days the diagnosis will usually clear

up. The second problem may be an insoluble one for several days. It is seen in those cases of typhoid fever already described as pneumo-typhoid or pneumo-typhus, in which the onset is characterised by all the symptoms and signs of croupous lobar pneumonia. This form of invasion is usually believed to be caused by the typhoid bacillus in the first instance selecting the pulmonary tissue, and sometimes the organism may be found in the rusty sputum almost from the beginning, in which case the diagnosis is established with certainty. When this cannot be demonstrated the case must remain doubtful till the Widal reaction is obtainable or the bacilli are found in the blood, urine, or stools, or till the first crop of rose-spots appears.

The third diagnostic problem is usually less difficult; it is met with when a complicating pneumonia supervenes at a later stage upon typhoid fever. The difficulty is practically confined to those cases of mild degree or to ambulatory typhoid, in which the patient may first seek advice for the complication, the primary disorder having been neglected. Sometimes in elderly subjects also the typhoid symptoms may be latent till the supervention of consolidation brings dyspnoea, lividity, toxæmia, and cardiac embarrassment into prominence. In all these conditions, however, some or many of the characteristic symptoms and signs of the primary disorder will be detected by a careful general routine investigation and a scrutiny of the history. Widal's reaction will be present, and the bacilli may be demonstrable in the blood, urine, or stools, if the presence of a rose-coloured eruption does not settle the diagnosis.

Appendicitis may be mistaken for typhoid fever, or the latter disease regarded as due to inflammation arising in the appendix, and often an operation has been undertaken for the relief of an appendicitis which has proved to be a case of typhoid fever. The chief points in the differentiation of appendicitis from typhoid are the marked localisation and the suddenness and violence of the attack of pain in the right iliac fossa, the circumscribed tenderness elicited over McBurney's point, the irregularity of the fever, and the history, which often reveals evidences of previous minor attacks suffered at longer or shorter intervals. The absence of all characteristic typhoid symptoms and signs and the presence of marked leucocytosis settle the diagnosis with certainty when moderate care is taken in the investigation of the case. Perforation of the appendix occurring in the late stage of typhoid fever is recognised by the symptoms of the secondary general peritonitis, which are practically identical with those caused by intestinal perforation.

Gastric catarrh in children affords a serious difficulty in diagnosis, especially when an epidemic of typhoid is present

in a warm autumnal season. If the catarrhal symptoms—nausea, vomiting, slight fever with or without constipation, and a markedly furred tongue—persist for several days the case may confidently be pronounced to be typhoid fever of the mild type, often styled '*gastric fever*,' which is identical with idiopathic infantile remittent fever. The eruption is very frequently absent in these cases, diarrhoea and tympany are more often absent than present, and the spleen may not be palpable; but Josias and Tollemer have shown that Widal's test is as valuable in the diagnosis of the infantile form as in the typhoid fever of adults.

Pyæmia is differentiated from typhoid fever, which it sometimes simulates, by the discovery of the primary septic focus, as an ulcerative endocarditis, acute necrosis of bone, &c., and by the blood-examination, which shows marked leucocytosis and characteristic features in the differential count; and Widal's reaction is never present, unless in the case of multiple infection or where the patient has previously suffered from an attack of typhoid fever. The same remarks apply to septicæmia, the *puerperal* form of which often bears a close resemblance to typhoid. The irregularity of the temperature-chart, as in the case of all tubercular infections, is a valuable diagnostic feature. The isolation of the exact micro-organism causing the septicæmia or pyæmia may often be effected by withdrawing 10 c.c. of blood from a vein in the arm under strict aseptic precautions. This is immediately diluted with a large quantity of broth (100 c.c. or more) and placed in an incubator; sub-cultures are daily made from this, by which means the causal organism can frequently be demonstrated. In practically the same manner the bacillus typhosus may be isolated after incubation of 10 c.c. of blood diluted by the addition of a still greater quantity of bouillon as early as the second or third day of the fever.

Trichiniasis has been often mistaken for typhoid fever, especially owing to its appearance in epidemic form, since the attack is ushered in with gastro-intestinal symptoms and fever, delirium, furred tongue, and sweating. The pain and tenderness in the muscles, with the characteristic œdema surrounding the orbits, the frequent respiratory distress, and the detection of the trichinæ in the stools are pathognomonic.

Prognosis.—The mortality differs widely in different epidemics, varying from 5 to almost 20 per cent. It is higher in hospital-practice, since the milder cases often are treated in the patient's homes. The death-rate has certainly of late years been considerably reduced by the routine use of hydrotherapy, but the mortality in the cases treated in the various hospitals under the London Asylums Board stands

at the high rate of 13·15 for 1905 and 16·02 for 1906. The mortality in war-epidemics is usually very high, as seen in the returns from South Africa in the late Boer campaign, where one in every five soldiers attacked by the disease died ; but, according to Osler, the mortality in the recent Spanish-American war only amounted to about one-third of this.

The general clinical features of the disease and the intensity or mildness of the fever often afford but little aid in forming an accurate prognosis in a given case, since death so frequently occurs from complications when least expected, as in perforation of the intestine, hæmorrhage, &c.

As a rule, the mortality is higher in women than in men, and the prognosis in children is decidedly more favourable than in adults, in whom the death-rate steadily rises after middle-life.

Obesity deepens the prognosis, and alcoholic subjects bear the prolonged fever badly. When these two conditions are combined, as they frequently are, a serious prognosis may be given from the onset. The mortality is high in the ambulatory cases, and it may be accepted that the risk of a fatal issue increases in proportion to the length of time during which the patient keeps upon his feet after the disease has first manifested its presence by malaise, &c.

Previous ill-health or debility appears to have little influence over the course of the disease, unless there have been structural lesions present, as disease of the heart and kidney, or unless an affection like diabetes has preceded the attack, since robust subjects are as likely to succumb as delicate ones. This is seen in the higher mortality amongst the robust rural new-comers into a district infected by the disease. High-livers have less chance of safely passing through a severe attack than the abstemious, independently of the use or abuse of alcohol in their dietary.

As to the special clinical features of the disease giving a clue to the ultimate prognosis in a given case, the following points may be stated :

When toxæmia is marked from the onset, independent of the intensity of the pyrexia and abdominal symptoms, as seen in the low character and persistence of the delirium, restlessness, insomnia, muscular tremors, and tendency towards a comatose state or coma-vigil as the disease progresses, the prognosis is grave, though sometimes a patient may recover after a prolonged period of unconsciousness preceded by marked mental symptoms.

Severe tremors are of even more serious import than severe delirium, and when the former occur with the mind clear most observers accept the view of Jenner that they may be taken as an indication of deep intestinal ulceration.

The mere intensity of the fever is of comparatively less importance, provided it does not approach hyperpyrexia, which always constitutes a grave condition ; but a prolonged high temperature during the second week, with only slight morning remissions, is always indicative of a severe attack. A sudden fall is often more serious than a sudden rise in the temperature, as the fall so frequently means either a smart hæmorrhage or a perforation ; whilst the rise is usually indicative of a less grave complication.

Severe abdominal lesions, showing themselves by excessive diarrhœa, sudden tympanitis, pain, melæna, or hæmorrhage, add to the gravity of the prognosis, and the latter complication by itself is a frequent cause of death. The former symptoms and signs deepen the gravity of the outlook, since they are often heralds of perforation or peritonitis, deep pain especially being indicative of an ulceration extending outwards to the peritoneal coat of the colon or ileum.

Epistaxis occurring during the third week, especially if associated with even trivial bowel-hæmorrhage or hæmaturia, is serious, as indicating a hæmorrhagic tendency ; and the same is true of petechiæ. Albuminuria is of no importance when it is but of the febrile degree ; profuse albuminuria, as in nephro-typhoid, is always a grave sign.

The scantiness or abundance of the eruption gives no aid in forming an accurate prognosis, and the same remark applies to sweating and the degree of splenic enlargement.

The state of the pulse is of importance as indicating the degree of cardiac failure in the later stages, intermittency, irregularity, great rapidity, and feebleness being grave features ; but as the intestinal symptoms are often no guide to the danger of perforation, so the state of the pulse sometimes fails to give warning of the sudden death which occasionally occurs from unexpected cardiac collapse. This latter complication or finale is much more likely to occur in men than in women, and may be witnessed during the third week or in the early stages of convalescence.

Perforation is almost invariably followed by a fatal peritonitis, the rare exceptions being when it happens to be of the pin-hole variety or when it occurs during convalescence from some error in diet, in which latter case life may be saved by operative interference. Splenic rupture is invariably fatal, but recovery has followed splenic abscess when diagnosed early and treated surgically.

Pneumonia is not necessarily fatal, though it adds to the gravity of the situation ; in elderly subjects either extensive bronchitis or pneumonia usually ends fatally. Pneumonia with latent physical signs, associated with cyanosis or dyspnœa, is nearly always fatal. In considering the influence of complications, the stage of the disease in which they occur is

often an important point in the prognosis. Thus a pulse of 120 is much less grave when the fever is high in the middle of the second week than when it occurs later on with a temperature which does not correspond. A pulse of 140 or upwards at any stage of the disease is nearly always a presage of death, which is not likely to be long delayed. A late pneumonia is usually more serious than an early one, being then liable to be of a mixed septic nature.

The prognosis in relapses is usually good unless perforation occurs or extensive and frequently repeated hæmorrhages appear, both of which complications are, however, very rare in relapses. Polyuria is believed to be a favourable symptom.

The prognosis in cases where the hygienic surroundings and nursing facilities are distinctly unfavourable should always be guarded; and here the choice must be made between the serious risks of removing the patient in the middle of his illness to a suitable hospital or of accepting the danger of treating him in his unsanitary environment. There cannot be a doubt of the increased risk to life in compelling typhoid-fever patients to undergo a journey to hospital of several miles when in the sloughing or ulceration stages of the disease, even where the most modern ambulance-facilities are provided, though some authorities maintain that the risk is not appreciably heightened by the length of the journey, provided the dangers of removal to and from the conveyance are skilfully minimised.

The prognosis in patients who have received preventive inoculations is better than in ordinary cases; the reports concerning the German soldiers employed in the late South-West African military operations show a typhoid mortality amongst the inoculated of only 4 per cent., contrasting with the death-rate of 11·1 in those not previously injected with the antityphoid vaccine.

The influence of pregnancy has been referred to upon page 1753.

Preventive Treatment.—This is of vital importance in a disease so widely distributed and of such common incidence. The methods to be employed are obvious from the study of the etiological factors. A pure water and uncontaminated milk supply being the most imperative, both these main sources should be scrupulously watched and guarded from the possibility of contamination. To check effectually the spread of the disease in a community compulsory notification has afforded most beneficial results by the facility which it gives for the tracing of the origin of the outbreak. During epidemics the water and milk should be boiled in every household as soon as the presence of the disease has been manifested in a district; shell-fish and salads should be prohibited as food, unless where the impossibility of their contamination is demonstrable; and precautions should be taken as far as

possible in the destruction of flies and other insect-carriers of the bacilli.

The disease being in the first instance due to the dissemination of the specific germs discharged in the excreta by typhoid-fever patients, in every case of the disease the most rigid disinfection must be effected of the alvine discharges and of the urine. The best routine disinfectant for all purposes, in the writer's opinion, is the free use of dry chlorinated lime, which should be added to the fæces and urine as soon as these are passed from the patient's body in the sick-room, and the mixture of the antiseptic and excreta should be permitted to remain for a time in the chamber before being emptied into the house-drain. The frequent flushing and thorough disinfection of the latter, of all closets, cisterns, cesspools, &c., should be systematically carried out. The discovery of the existence of *chronic typhoid-carriers* is mentioned upon page 1737.

As regards the urine, the fact has already been emphasised that it may contain the bacilli in almost pure culture for many months after convalescence; and to this fact perhaps more than to anything else is due the difficulty in stamping out the disease. The routine administration of urotropin has been advocated during and after convalescence; but of more effect is the impressing upon the mind of the typhoid convalescent the criminality of permitting soil, clothing, water, utensils, and every other conceivable medium of the spread of the disease being polluted by his urine.

The serious question of the necessity for the rigid isolation of the typhoid patient during his illness has been already incidentally referred to and the custom of treating the disease in the open mixed wards of a general hospital condemned. There can, however, be no doubt of the possibility of this being done without danger to others, provided every precaution be taken in the immediate destruction of all germs in the excreta, sputum, &c., and in the thorough disinfection of all body-linen, bed-clothing, drinking and other utensils. But the possibilities of some trifling neglect or unconscious omission of antiseptics are so numerous that danger should be always feared; thus reference has been already made to the spread of the disease through the use of soiled enema-syringes and clinical thermometers: even the bath in which the typhoid-fever patient receives his antipyretic treatment may convey the disease to others unless carefully disinfected. The remnants of food unconsumed in the sick-room, as beef-tea and milk, should be poured into the chamber along with the disinfectants employed for the purification of the excreta. The dangers of nursing typhoid-fever patients in a general ward are greatly increased in those grave cases where the diarrhoea is frequent, and especially when the motions are passed involuntarily and cannot be disinfected.

Preventive inoculations have been extensively practised by Sir A. Wright, Semple, and others amongst the English soldiers in India, South Africa, and other military stations. The vaccine-material consists of virulent cultures in bouillon of the bacillus typhosus destroyed by heat. The dose, according to Wright's method, should consist of about one thousand millions of the sterilised bacilli, followed in fourteen days by a second injection of about twice this number. The inoculation causes mild constitutional symptoms and some local reaction, showing itself in tenderness and slight swelling. Wright claims for this operation a reduction of 50 per cent. in the incidence of the disease and a similar or somewhat greater diminution in the mortality amongst the inoculated who subsequently suffer from typhoid. In the German statistics already quoted, which are the latest available, the mortality in the disease was reduced to almost one-third of that amongst the uninoculated soldiers, and the most severe cases of the disease amongst the inoculated subjects were found to occur in those who had been but very recently subjected to the antityphoid vaccine. The modifying influence of the inoculations appears to last for a period of about a couple of years, and the method promises to be of great value in conditions like those which prevail in military operations where large bodies of men are liable to be submitted to infection. In the Boer war, owing to the unusual prevalence of flies, sand-storms, and a very vitiated water-supply, the value of the preventive inoculations was less obvious than in the Indian stations.

General Treatment.—Though no known drug has been found to exert any specific action upon the bacillus typhosus or its toxins in the human body, nevertheless there is scarcely to be instanced any other disease in which skilful management has such a wide scope and beneficial effect. The subject is a large one owing to the numerous complications and phases through which the patient must pass in his prolonged struggle. The author has detailed the different methods of dealing with the affection in the fourth edition of his 'Dictionary of Treatment,' on pages 965 to 985, and only the more important points need be here referred to.

The patient is put to bed at the earliest possible moment in a large well-ventilated room, a hair-mattress upon the top of a woven wire one being selected; the bed-clothing should be light, and two small movable beds are very desirable, when possible, so that the patient may have the advantage of a total change of bed-linen at times with the minimum of disturbance. The urinal and bed-pan are indispensable, so also are draw-sheets and mackintoshes in most cases. A matter of very great importance in the use of the bed-pan should be noted. Some patients, especially males, cannot

use this nursing utensil without serious difficulty and great straining; and though it is very desirable that the patient from the outset should have his bowels relieved without getting out of bed, the choice of evils must be balanced, as undoubtedly grave danger of perforation occurs from a cast-iron order by the physician that the patient under no circumstances whatever should be permitted to get out to the night-chair. Sometimes it may be necessary that he be instructed to pass his motions in the bed into tow, flax, or oakum.

Dietary is the most important element in the treatment and management of an attack of typhoid fever from the onset till convalescence is well established. The food should be liquid, and all the requirements of the case are usually met by a generous but not too lavish amount of pure cow's-milk, alternated at times, when diarrhœa is not profuse, with clear soup, beef-tea, beef-jellies, or strained mutton-broth from which all traces of fat have been removed. Many patients can take their milk undiluted, whilst others require the liquid to be mixed with an effervescing water. Sometimes, when gastric disturbances are present and where curd appears in the stools, peptonisation of the milk may be demanded.

Ewart, who advocates what he calls 'the empty-bowel treatment,' insists upon a dietary which leaves the minimum of intestinal residue, with which intention peptonised whey with egg-albumin added to the milk previous to the peptonising process is administered. He states that two and a-half pints of this whey, to which about a drachm of salt is added with the yolk of one egg, in addition to plenty of water, are quite sufficient daily to meet all the demands of nutrition in the early stages of the disease. There are good reasons for believing that too much milk is often administered, and it may be accepted that about three pints in the twenty-four hours are quite sufficient in most cases to sustain the demands made upon the patient's strength. Abundance of water should be always permitted; where this is forbidden the patient uses more milk to assuage his thirst than his digestive apparatus can assimilate. Of even more importance is the necessity for administering the milk in small and frequently repeated quantities; a large wineglassful every couple of hours will represent between two and three pints daily, allowance being made for larger doses after sleeping. The inspection of the stools will afford evidence of whether this amount is being digested by the patient.

Eggs are recommended in the dietary by many physicians, but their administration should never be forced upon a patient when the intestinal disturbance is severe, unless in the harmless form of peptonised egg-albumin. Arrowroot,

gelatin, isinglass, or any form of animal-jelly may be permitted when the bowels are loose, and a fruit-jelly or strained mutton-broth when there is constipation; cream is sometimes relished. The serious question of a return to solid food is to be met with in the early convalescing stage. Periodically the tradition is challenged that solids should never be given throughout the disease until after an apyrexial period of fourteen days. The revolt against this is usually reported as successful till a case of perforation or hæmorrhage traceable to the indiscretion in diet occurs, after which the physician returns to the old-established practice and never again departs from it.

In connection with the dietary the important question of giving alcohol arises. There should never be a doubt in the physician's mind when called to treat a patient in typhoid whose daily habit has been to take alcohol in some form; the stimulant cannot safely be withheld. Even if he has been in the habit of taking more than is safe, when overtaken by an attack of disease it is not the time to begin a reformation with the dangers of a prolonged continuous fever confronting him.

Under other circumstances in an ordinary smart attack the generally accepted routine practice is to omit alcohol in every form from the dietary, unless in the presence of some serious complication which demands a rapidly acting cardiac stimulant. The indications for and against the use of alcohol are discussed in the writer's article previously referred to. The chief symptoms calling for its exhibition are weakness, irregularity and compressibility of the pulse, and the drug may be given in full doses as long as its obvious action is to bring the patient nearer to the normal, as forcibly stated by Sir Lauder Brunton. The withholding of alcohol in all cases of the disease seems to the writer to be a practice as much open to serious objection as is the indiscriminate routine use of it in every case. Either method gives a higher death-rate than the judicious administration of the agent in selected cases when the heart requires it, though often very unpromising cases pull through without its aid. Whisky or brandy should always be preferred to wine, and very rarely should a daily allowance of ten ounces of either be exceeded.

Of routine treatments of the disease, that by purging is sometimes advocated, and recently it has been strongly commended by Thistle with the view of effecting elimination of toxic products in the intestines. The same objection holds more forcibly to this routine than to the alcoholic. Purgatives are undoubtedly valuable in some cases and lethal in others. When a cathartic is required a mild dose of castor oil answers all purposes where a copious washing-out of the colon by a tepid enema is not feasible. Large doses of

calomel, except perhaps once at the beginning of the attack, are to be condemned, as are all agents which are liable to set up an exaltation of the peristaltic action.

More reasons can be advanced in support of another routine method—viz. the administration of so-called intestinal antiseptics; these as a rule can do little harm, unless calomel in full doses be used with this intention. As pointed out by Goodall, the human intestinal canal cannot be treated as in a test-tube experiment, and any solution capable of destroying the germs in it must affect injuriously the mucous membrane. Moreover, a general invasion of the blood and entire tissues of the body has already occurred; nevertheless, intestinal fermentation and decomposition may be kept to a certain extent in check and tympanitis may be ameliorated by the administration of such agents as oil of cinnamon, naphthalin, turpentine, salol, chlorine, &c., or by giving powdered charcoal in association with petroleum, as advocated by Ewart in conjunction with his empty-bowel treatment. Dr. W. Calwell reports favourably of oil of cinnamon.

Hydrotherapy.—Routine administration of drugs of the antipyretic class is universally condemned; the only one which is not positively injurious in large doses is quinine, and this agent, except in the case of children, has very little effect in favourably influencing high temperatures. The only reliable antipyretic in typhoid fever is tepid or cold water, which may be used in various ways; when employed as a routine treatment of this continuous fever the method is frequently referred to as Brand's Treatment.

Authorities differ greatly about the limit of the temperature which is to be taken as an indication for hydrotherapy, but an average may be accepted of between 102.2° (Brand) and 103° (Liebermeister). When this degree of pyrexia is reached the patient receives a bath at about 75° F. for fifteen to twenty-five minutes every three, four, or six hours, and the method may be employed throughout the entire period of the fever. Lower bath-temperatures are seldom indicated except in dangerous hyperpyrexia. Some physicians prefer to place the patient in a bath of 90° or 95° , and cool it down rapidly by the addition of cold water or ice till 65° is reached. The method has been modified by Barr, who advocates the treatment of the patient by prolonged or continuous immersion in a tank of water kept at a temperature of about 95° .

The rectal temperature is taken in the bath, and when the mercury falls $2\frac{1}{2}^{\circ}$ the patient is removed to bed, and a little hot stimulant administered. It is claimed for the hydrotherapeutic treatment that it not only effectually controls the fever, but increases the blood-pressure, reduces the frequency of the pulse-rate, diminishes or obliterates diastolic murmurs, and has a very favourable influence over the symptoms due to

general toxæmia, often inducing sleep and stimulating the renal and respiratory functions. The statistics prove that when the method is used as a routine and not merely resorted to in spasmodic fashion for the relief of very high temperature the death-rate has fallen to almost half the usual percentage.

The objections to and difficulties in carrying out this plan of treatment in private practice are obvious; it requires a staff of skilled and experienced nurses and necessary equipment and nursing appliances not usually available outside hospitals. The main danger arises from the possibility of injury to the intestinal lesions in the mechanical removal of the patient from his bed to the bath and from the latter back to bed again. It is clearly contra-indicated in severe hæmorrhage, deep abdominal pain, or other evidence of severe intestinal ulceration or peritonitis, or when phlebitis is present; but bronchial or even pneumonic troubles usually are favourably influenced by it.

Most, if not all, of the benefits of the bath may be obtained by constant sponging of the body with cold or iced water without entailing any danger in moving the patient from his bed. This plan, to be effectual, requires most patient and conscientious efforts upon the part of the nurses. Where prejudice against the use of cold water has to be combated, tepid sponging of the different parts of the body in detachments must be assiduously carried out. The cold pack can always be substituted for the bath when all movement of the patient's body is contra-indicated and when the temperature is very high, the body being enveloped in a sheet wrung out of tepid water over which cold water may be made to trickle for a quarter to half an hour if necessary in hyperpyrexial conditions.

Guaiacol painted over a considerable area of the skin brings down the temperature usually with certainty and rapidity, but it should never be resorted to owing to the dangers of serious collapse liable to follow its use, and the same remark applies to all the newer internal antipyretic agents. Moreover, the rebound upwards after the temporary fall is often greater than the temperature which these agents are employed to reduce.

Serum-therapy still awaits discovery which will render an antityphoid serum efficient in the treatment of the disease. The results obtained by Allan Macfadyen in obtaining an *Anti-endotoxin* by the intravenous injection into goats of a typhoid endotoxin obtained by triturating the bacilli at a temperature of liquid air afford some hope that the therapy so successful in the treatment of diphtheria may be soon applicable in typhoid fever. The results of treatment by the serum prepared by Chantemesse, Bokenham, and others are also promising, but not convincing.

The treatment of an uncomplicated attack of the disease is therefore mainly a matter of dietary and nursing ; if the patient or his friends insist upon his having drug-treatment, the old Swedish routine of 15 mins. of dilute hydrochloric acid thrice daily in a wineglassful of water never does any harm, and usually assists in keeping the tongue less furred ; and as it supplies the free acid in conditions where there is often a diminished supply of it in the stomach, it usually is beneficial.

The treatment of symptoms and complications as they arise calls sometimes for active drug-administration, and by the judicious employment of therapeutic agents life may be often saved. This remark applies chiefly to intestinal hæmorrhage, in which sometimes a fatal issue may be averted by prompt dosage with chloride of calcium supplemented by hypodermic or intravenous injections of warm saline solution. The drenching of the patient by vegetable astringents, as ergot, tannin, turpentine, or by lead and copper salts, with the view of checking hæmorrhage or profuse diarrhœa, only diminishes his chance of recovery by arresting the digestive functions.

Where the diarrhœa is excessive tannalbin may be combined with morphia in moderate amount ; and constipation is best treated by washing out the colon or giving a small dose of castor oil by the mouth.

Abdominal pain and tympanitis will demand warm or hot turpentine stupes, and possibly morphia hypodermically ; the use of the long tube for the reduction of abdominal distension is nearly always a futile procedure, though frequently recommended.

Perforation is so absolutely hopeless, if left alone, that to treat it by the expectant method is to deprive the patient of the chance which laparotomy affords. Promptness of action is demanded, and the fatal mistake is commonly made of waiting till the supervention of unmistakable evidence of peritonitis, with the view of clenching the diagnosis before deciding upon operation. Peritonitis of more or less severe degree has already occurred before perforation is usually suspected ; by modern physicians the first symptoms of perforation are now recognised to be those produced by the resulting peritonitis, and are not due to the mere rupture of the intestinal tube at the point of deepest ulceration. Hence the necessity of immediately performing laparotomy ; and, though the operation is often fatal, numerous lives have been saved of late years even when the condition of the patient seemed to be practically hopeless. The outlook is much brighter after operation for perforation following an indiscretion of diet in the stage of convalescence, and the circumstances must be indeed rare which would justify the

surgeon in refusing to open the abdominal cavity in such a case.

Delirium and insomnia may be relieved by the ice-cap; cardiac weakness and collapse call for strychnine hypodermically and alcohol in full doses by the mouth or bowel. The general toxæmic symptoms often yield to hydrotherapy, and are usually intensified by dosage with morphine or other narcotics. Bed-sores should very seldom be witnessed when ordinary care in the nursing has been exercised; when they do occur they must be treated by antiseptic applications directed by recognised surgical principles and by frequent changes of the patient's position, or by the use of a water or air bed. Abscesses in the cellular tissue or under the periosteum require free evacuation, and phlebitis should always contra-indicate the slightest movement of the limb in which the thrombosis has occurred. The necessity for close attention to the state of the bladder cannot be too frequently emphasised; life is often jeopardised and sometimes lost by neglect of ordinary watchfulness in this matter, and strict sterilisation of the catheter is demanded immediately before the instrument is used. Though there is little necessity for drugging the patient throughout his attack with urotropin when the urine is found to contain the bacilli in great numbers, as recommended by some, still it is a good rule to give 10 grs. twice a day in all catheter-cases to diminish the risks of septic infection by the instrument when the operation of passing it must be trusted to unskilled hands.

The treatment or management during the convalescent stage of the disease is sometimes quite as important as that of the active stage in ordinary cases. The dietary should be regulated with great care, and it is a wise and necessary rule never to permit solid food to be taken till the temperature has been normal in the evenings for a clear fortnight. The physician should not yield to the importunities of a hungry patient; the latter should be assured that all the demands of his case can be fully met by a generous supply of liquids and soups thickened by smooth farinaceous preparations like arrowroot, cornflour, or oaten flour. These latter may be permitted freely of the consistence of a stiff porridge or of a semi-solid blanc-mange. Fruits are at this period dangerous, especially in the case of children, who are apt to bolt orange-pips and grape-stones when the appetite is ravenous, as it often is during convalescence.

The first period of getting out of bed should be regulated by the total length of the attack and the severity of the fever. In mild cases only should the patient be permitted to leave his bed for a few hours at a time within a week after the cessation of the fever. Where there has been clotting in the veins of the lower extremity extreme caution should be

exercised in keeping the patient in a state of absolute rest for a prolonged period of several weeks. Close attention should be paid to the state of the bowels, and constipation must be strictly guarded against. Upon the first appearance of a relapse the patient must be immediately sent to bed, and all the precautions indicated in a severe attack of the disease should be enjoined.

The treatment of such sequelæ as peripheral neuritis and typhoid spine is to be carried out by prolonged rest and, at a later stage, by gentle massage, electricity, &c., as are indicated in the case of alcoholic or post-diphtheritic multiple neuritis.

TYPHUS FEVER

This is an acute and highly contagious disease, usually appearing in epidemic form, and characterised by a sudden or definite onset, with marked nervous symptoms and the appearance of an almost pathognomonic exanthem about the fifth day, the disease tending towards an abrupt defervescence by crisis, usually upon the fourteenth day.

Etiology.—The disease has been known from the earliest times as constituting many of the most serious epidemics in periods of war, famine, or other form of destitution. Its older names of *military*, *camp*, *jail*, *ship*, *pestilential*, and *putrid* fever imply the recognition of the important etiological factor of overcrowding or deficient ventilation associated with want or destitution. Owing to improved hygiene the disease has now become one of the rarest, even in Ireland, where in many districts it has lingered in a sporadic form till a quite recent period. Though the affection affords one of the most typical examples of a truly epidemic disease, it is remarkable how isolated instances crop up sometimes which cannot possibly be traced to infection from an individual case. One such of the most perfectly typical description under the writer's care occurred in an individual many years ago who had not been out of his circumscribed district for many months, though no case of typhus was known in Ireland at the time; and the writer himself suffered from the disease long afterwards under similar mysterious circumstances, no case being known as existing for a year or more within the possible zone of his environment. Instances of such isolated examples of the disease led Murchison to believe in the spontaneous-generation theory of typhus, which is now universally acknowledged to be absolutely untenable. Murchison held that the poison causing the disease developed *de novo* under conditions of overcrowding and squalor; the two isolated cases just referred to occurred under the most ideally perfect opposite conditions and spread to no other individuals.

There cannot be a doubt that the etiological factor in typhus is a specific microbe; though many organisms have been described from time to time, the specific bacillus still awaits demonstration. It is possibly much more widespread in its distribution than is recognised, and probably may lie dormant or harmless for many years till its virulence becomes established by the faulty hygienic conditions of overcrowding and imperfect ventilation heightened by destitution. The tenacity with which the microbe clings to life in certain isolated areas has been demonstrated by the numerous outbreaks in the small island of Aranmore, in one of which during 1902 Dr. William Smith lost his life in his heroic effort to save the fever-stricken islanders in a manner which thrilled the inhabitants of Great Britain.

The specific virus is given off in the expired air and in the exhalations from the patient's body, and probably also in the excretions from the bowel and kidney. From these sources it is conveyed in the air by means of fomites, and finds its way into the healthy body. It is readily conveyed in articles of clothing, bedding, and furniture, and lingers in the crevices of woodwork and the cracks of walls, lasting longer in and, it is said, conveyed more effectually by, dark-coloured fabrics. As pointed out by Sir John Moore, who has had unusual experience of the disease, the striking-distance of the typhus-germ is not great, provided only a fresh-air space separates the healthy from the sick, the poison being readily oxidised; hence, unlike the contagia of small-pox and scarlatina, it does not cross open spaces. It is probably destroyed readily by direct sunlight, and clothing is effectually rendered sterile by a dry heat under the boiling-point of water. The virus is volatile and lighter than the ordinary atmospheric air—an important point in the prevention of the disease, which should be treated when possible in the upper rooms of hospitals or dwellings. The period of greatest danger of contagion does not occur till about the end of the first week.

Though highly contagious under conditions of deficient ventilation, from the above facts it becomes evident that with ample air-space, a free current of pure air, and abundant sunlight, the virus is materially robbed of its infective qualities.

All ages, both sexes, and persons in every condition of life are liable to suffer in times of epidemic, but the disease is chiefly met with amongst the poor and destitute between the ages of ten and forty-five; rarely is it conveyed to the healthy by a third person. From the nature of their occupation, obviously nurses, medical men, and ward-attendants suffer heavily in times of epidemic, and laundresses very frequently catch the disease from contact with soiled linen. The predisposing causes are those seen in operation in most other infections, as previous debility and diseased conditions;

fatigue (bodily or mental), alcoholism, idiosyncrasy, and the condition of body induced by imperfect aëration of the blood, as seen in overcrowding and imperfect ventilation independent of the actual proximity of a typhus patient, are powerful predisposing causes. The popular idea that the dread of catching the disease renders an individual more susceptible may contain a germ of truth. New-comers are especially susceptible to the disease when settling in a typhus district.

One attack generally confers a life-long immunity to the disease.

Morbid Anatomy.—There cannot be said to be any characteristic lesion discoverable after death save the eruption and the petechiæ in association with it, both of which are obvious at the autopsy. The blood, tissues, and internal organs show the changes always found after death in conditions associated with continuous and high pyrexia. The heart is always found softened, and sometimes remarkably friable; sometimes it is obviously dilated; the muscular fibres show advanced fatty degeneration unless death has occurred at an early stage. The blood is fluid and almost black in colour, and any clots found in the heart are soft or diffuent, fibrin being recognised with difficulty in them. Minute hæmorrhagic extravasations are common upon the surfaces of the internal organs, which are always deeply congested. The spleen is almost invariably enlarged, soft, and quite friable; the liver shows similar changes to a less extent, and the kidneys are swollen and sometimes exhibit the tubal lesions characteristic of acute Bright's disease. Pulmonary and bronchial congestion are present; the bases of the lungs always show signs of hypostatic congestion; and sometimes marked meningeal engorgement, with the effusion of serum within the cranium is obvious. Rigor mortis comes on rapidly and is of short duration, and the stage of putrefaction supervenes with remarkable rapidity, though usually the body does not show a marked degree of emaciation, unless death has occurred after the crisis.

Symptoms.—The *incubation-period* varies considerably; it is held by some authorities that a concentrated dose of the poison may act so swiftly as to leave practically no appreciable interval between the exposure and the onset. As a general rule, twelve days may be accepted as a working average.

The *invasion* is nearly always sudden and abrupt, being not rarely ushered in by one marked and prolonged rigor; where this is absent a very obvious series of chills are complained of, the patient feeling as if cold water was being poured down his spine; malaise, languor, muscular pains, and intense headache speedily follow or accompany the chilly sensation. There is total anorexia, and often a feeling of nausea, but seldom actual vomiting occurs, and the tongue

is covered with white fur. With the headache, which is frontal, there is commonly a sensation of distension within the cranium, and giddiness or tinnitus is present. The pulse begins to increase in frequency from the start, and the temperature rises steadily, so that the fastigium may be reached by the fourth evening, when the mercury may register 103° to 105° , or even more.

The feeling of prostration is so acute that the patient may be compelled to take to bed upon the first day ; very generally he is knocked off his feet within forty-eight hours after the first sensation of chilliness.

At this period his face is congested and dusky, his eyes suffused, and the edges of the eyelids appear somewhat swollen ; the entire expression, even at an early stage, becomes indicative of heavy dulness, listlessness, and languor, so that the patient appears usually at a glance to be more obviously ill than at the corresponding period of a typhoid attack. When he falls asleep (insomnia is always more or less marked) his brief slumber is apparently disturbed by dreams which confuse his first waking moments. This condition is the forerunner of delirium, which is often marked as early as the fourth or fifth day, though the patient can usually be made to pull himself together for a time and answer questions sensibly.

The delirium of typhus is an almost constant feature of the disease ; the writer has only once seen it absent in severe cases. It is often of an active type (*delirium ferox*) resembling acute mania, and when muscular tremors appear later there is a semblance to delirium tremens. A fixed delusion all through the delirium may occupy the patient's mind : he fancies sometimes he is being pursued, and he makes desperate efforts at escape, requiring a staff of attendants to keep him from jumping out of bed or flinging himself through the window. This form of delirium is sometimes well established at night before the end of the first week, whilst in the mornings the wandering may show itself in a low muttering, indicative of mental exhaustion. The abdomen is flat ; tympanitis, tenderness, and gurgling are absent ; the spleen can usually be felt somewhat enlarged.

Eruptive Stage.—The appearance of the rash late upon the fourth evening or by the middle of the fifth day usually coincides with the development of the mental and nervous excitement, which produce the dissipation of the initial severe headache.

The eruption first appears usually about the folds of the arm-pits and upon the back of the wrists, and soon afterwards upon the sides and front of the abdomen, and within forty-eight hours the general cutaneous surface is fairly covered, though the face and neck usually are free from it.

It is generally described as a *mulberry* exanthem ; sometimes, though rarely, it is preceded by a scarlatiniform roseolar eruption. The fully developed eruption is always distinctly *macular*, consisting of numerous spots of irregular outline and shape, each of a dull red or mulberry colour ; the largest may measure as much as the third of an inch in diameter, and they may show a tendency to confluence. The smaller spots *at first* differ little in appearance from the typhoid papules, as they are of a brighter red than the mature rash, and may be felt by the finger as slightly elevated above the cutaneous level, and may disappear upon pressure. Within twenty-four, or at most forty-eight, hours, these latter characters disappear, and the spots remain as small, livid, or dingy-red roundish maculæ, whose margins blend imperceptibly with the surrounding skin, showing no elevation and remaining uninfluenced by pressure, because by this time the spot has become a true petechia or small ecchymosis, as pointed out by Fagge, owing to the escape of blood from the vessels into the substance of the cutis. Sir John Moore emphasises the fact that the rashes of other diseases may be *accompanied* by the development of petechiæ, but are not, like the macular rash of typhus, *converted into* petechiæ. The eruption comes out completely within about thirty-six hours of the appearance of the first spots on the wrists or chest, and it remains till after the crisis or after death. There is never a second or successive crop, as happens in typhoid fever, after the first crop of papules fade. An important feature of the fully developed rash of typhus is the so-called *sub-cuticular mottling*, which consists of a fainter and duller reddish mottling, as if caused by a more deeply situated macular eruption, visible beneath the cutis. This combination of distinct maculæ with sub-cuticular mottling constitutes the pathognomonic mulberry eruption of typhus.

The degree of petechial hæmorrhage varies, sometimes the eruption becoming livid or purple ; and the severity of the attack may be usually gauged both by the copiousness of the eruption and by the rapidity of its tendency towards lividity. The petechiæ at this stage often resemble, and have been sometimes mistaken for, a crop of flea-bites, but the central punctum is always absent. Rarely do larger purpuric spots or vibices complicate the eruption. A peculiar and characteristic, unpleasant, or even cadaverous odour is usually exhaled from the skin ; sweating and sudamina are often present.

From the period of the full development of the rash onwards till the crisis the symptoms and signs deepen, the active nervous excitement gradually giving way to manifestations of nervous and physical prostration, as the delirium assumes more of the muttering character, and the patient tends to

pass into the so-called 'typhoid state,' a phrase first applied to this stage of typhus and not to the condition seen in typhoid or enteric fever. In grave cases liable to end fatally this state is usually well marked. The patient lies listlessly upon his back, sinking downwards from off the pillow muttering incoherently and incessantly, usually with his eyelids closed over his suffused or injected eyeballs, with their pin-hole pupils. His face is congested, the moist white fur having disappeared from his tongue, which is now dry, brown, hard, and crusted, and sordes accumulate upon his teeth and lips. The stupor is intensified by deafness, which is always marked; and should the state of coma-vigil, with its open staring eyelids, be present, it is obvious that he only hears when shouted to. Tremors become better marked; subsultus, twitchings, fumbling or picking at the bed-clothes (floccitatio and carphologia) are seen, and he gradually passes into complete stupor or coma. In less severe cases the fever and active delirium continue without the graver toxæmic symptoms becoming marked, till the appearance of the crisis, after which all suddenly disappear.

The pulse at this stage is always quick and feeble, and often no marked dicrotic feature is noticeable in it throughout; in cases which terminate fatally it may register 140 early in the second week. The heart soon shows signs of weakness, some diminution of the force of the apex-beat being perceptible about the period of full development of the eruption; the first sound nearly always exhibits feebleness, and in severe cases may be inaudible; more frequently the foetal characteristics of both sounds become obvious.

The respiration-rate in ordinary cases is quickened in proportion to the pulse-acceleration, but in grave cases considerable variations from the normal ratio are observable as sighing, cerebral, or Cheyne-Stokes respiration develops.

The *temperature* in typhus, as already stated, rises rapidly, so that it usually reaches its height by the fourth evening, but sometimes this limit has been reached before the end of the first twenty-four hours; in other cases the fastigium has not been arrived at till the seventh or eighth day. A maximum temperature above 105° or 106° is comparatively rare. During the second week a fall or remission about the eighth or ninth day is very commonly observed, and has been called the pseudo-crisis, after which the second fastigium is usually reached by the eleventh day, from which period the evening pyrexia often remains steady and high till the final fall, ending in the true crisis.

In grave cases there is no remission about the eighth day, and the fever continues to rise during the second week, when it may culminate in hyperpyrexia. The evening elevation of temperature all through may be high without causing serious

anxiety to the physician, as long as there is a fair remission or drop on each morning, which causes a series of breaks in the continuity of the pyrexia. The absence of these morning drops during the second week, or the presence of a sudden evening rise, is very liable to be caused by some pulmonary complication.

Hypostatic congestion of the bases of both lungs is so constantly found during life and after death in fatal cases that Murchison regarded it as a feature of typhus and not as a complication; it may exist without symptoms of cough or expectoration in the second week, when the patient continues to lie helplessly upon his back.

The urine exhibits the usual characters met with in pyrexial conditions; it is concentrated and often contains traces of albumin, and the chlorides are markedly diminished; retention is common.

The bowels are usually constipated throughout, though sometimes diarrhœa sets in during the second week; vomiting is comparatively rare. In the typhoid state both urine and fæces are usually passed unconsciously, even in cases which end in recovery.

The Crisis.—The defervescence is often described as precipitous; it occurs most frequently upon the fourteenth day, rarely more than one or two days earlier or later. The first sign may be a marked fall in the temperature, when this is taken at short intervals about the expected period of the crisis. Not unfrequently the thirteenth day may witness a rise of one or two degrees several hours before the crisis commences. The patient falls asleep, and after some hours may waken up and become calm and collected after a few minutes' confusion of thought and vision. There is often profuse sweating, and a large quantity of urine is passed; less frequently diarrhœa accompanies the phenomenon. The drop in the temperature may exceed 5° or 6° F. during twelve hours or even less; in other cases the crisis is less precipitous, the fall being spread over twenty-four hours or longer; there may be a slight rise upon the succeeding evening, but generally the thermometer continues to register the normal from the initial fall and onwards. Every symptom fades with the disappearance of the pyrexia: the pulse falls rapidly and completely to the normal rate, the tongue cleans, and the nervous symptoms vanish likewise; so that within twelve to twenty-four hours the patient may be safely regarded as convalescent, and the appetite becomes ravenous.

The presence of a complication may delay the crisis till the seventeenth day, in which case the defervescence is likely to be less abrupt or sometimes to occur by ordinary lysis.

When the typhoid state has been well-marked, with subsultus, coma-vigil, profound stupor, &c., if the patient has

survived till the usual critical period, no crisis occurs, and he succumbs to asphyxia or cardiac failure, or collapses under the intense toxæmia. More frequently in fatal cases death takes place before the end of the second—rarely during the first—week with the eruption showing much lividity. Nearly always some hours before death there is a marked agonal rise of temperature, amounting to several degrees, in which hyperpyrexia is reached.

During epidemics in crowded communities, where the hygienic surroundings were highly unfavourable, the type of the disease known as *Typhus Siderans* has been not unfrequently met with, in which death has occurred during the first three or four days, or earlier, before the eruption had time to appear. In ordinary epidemics the opposite condition is not rare, cases running a mild course with but a febrile temperature, and ending in convalescence by the end of the first week.

In children under ten years of age the eruption is often scanty and sometimes entirely absent, though occasionally it is profuse and closely simulates the rash of measles, and invades the face. The symptoms as a rule are much milder, the disease runs a shorter course, and the mortality is very low.

Relapses are exceedingly rare, or almost unknown, in contrast to the tendency so frequently observed in typhoid fever.

Complications.—These are uncommon if we exclude hypostatic congestion and cardiac weakness, which should rather be regarded as features of the disease. Acute pneumonia is the most frequent and important of the complications; it may be of lobar or lobular type, basal or apical, setting in during the first week or towards the end of the second, or it may appear as a sequela, showing its presence some days after the crisis.

Bronchitis is of less gravity, and some degree of bronchial catarrh appears to be almost as constantly present as in typhoid fever. In adynamic cases, however, the bronchial complications may prove fatal, when the patient towards the middle of the second week becomes physically unable to bring up the viscid secretion from the tubes. It is in this condition that grave hypostatic congestion is so liable to supervene upon the bronchitis. Gangrene of the lung has been often observed to follow attacks of the disease occurring amongst surroundings of unusually severe destitution, probably as a sequela to pneumonia.

Pleuritis is rare, and when present is liable to be purulent. Phthisis, though included amongst the complications of the disease, is probably due to the lighting up of an old latent tubercular focus. The laryngeal complications differ little

from those met with in typhoid, the most dangerous condition being acute œdema of the glottis. Thrombosis of the leg-veins, producing a condition allied to phlegmasia alba, and in which the lymphatics are probably also involved, has been many times met with in convalescence, and arterial thrombosis or embolism has been followed by a true pyæmia, involving the joints, lungs, liver, spleen, and other viscera in metastatic abscesses.

Hæmorrhages into the skin or from the mucous surfaces, due to a purpuric or scorbutic condition of the blood, intensified or caused by the previous destitution, were common in the Crimean campaign.

The fatty degeneration of the heart has been already dealt with as an almost constant feature of the disease, but endocarditis and pericarditis are very rarely met with as complications or sequelæ.

Many epidemics have been characterised by the frequency with which local gangrene has attacked the cheeks (*cancrem oris*), the nose, ears, fingers, toes, and genitals. Hospital gangrene was very common in the camps amongst the wounded soldiers affected by typhus in many former epidemics. Bed-sores are much more frequent in typhus than in typhoid, notwithstanding the much shorter duration of the fever, their presence being due to the more profound depression of the nerve and spinal centres. Parotitis and bubonic swellings were formerly very prevalent in some epidemics.

The complications affecting the nervous system appear as intensifications of the profound toxæmia affecting the cerebral tissue. Rarely is true meningitis present, though the cerebral symptoms often arouse a suspicion or conviction of its presence. Hemiplegia, with or without aphasia, occurs very rarely, and is probably due to embolism. Various localised paralyses, due to peripheral neuritis arising during convalescence, are not uncommon.

Mental failure, mania, and other forms of psychosis are rare; occasionally, as in typhoid, they occur during convalescence, and generally pass away after some months, leaving the patient's cerebral powers unimpaired.

Jaundice is a rare complication, and sometimes proves fatal. Kidney-disease very seldom follows an attack of typhus, though albuminuria is very common during the height of the fever. Cystitis may follow retention of urine and septic catheterisation.

The writer experienced a serious complication in his own person, which he has not observed recorded by others. The fourth costal cartilage broke with a distinctly appreciable dull snap or thud upon the left side during the twelfth or thirteenth day of the disease, in making a severe expiratory effort; considerable pain and effusion followed, and the

formation of a false joint resulted, which caused great suffering for more than a year, but it ultimately yielded to prolonged rest and fixation. Such a complication shows the profound tissue-change which the toxins are capable of inducing, though no other evidence save a tachycardia of several months' duration manifested itself.

Other diseases have been observed to follow immediately after the cessation of the fever, and in rare cases during the attack, as erysipelas, diphtheria, measles, scarlatina, small-pox, typhoid fever, purpura, and scurvy.

The convalescence after an attack is usually uninterrupted and progressive, but considering the short duration of the disease, the profoundness of the muscular debility is often most striking, the patient sometimes being unable to stand at a period when a typhoid-fever convalescent is often able to walk about without difficulty.

Diagnosis.—In the presence of an epidemic there is seldom a difficulty in the recognition of the disease, even before the appearance of the eruption, since the abruptness of the onset, high fever, and marked physical and mental prostration, accompanied by the characteristic dull heavy expression of the face, with suffusion of the conjunctivæ, are scarcely to be mistaken. Sometimes, however, the depression of the onset may be counteracted by a strong physique and firm will, as seen by the writer, who was called some years ago to see an elderly surgeon who had fainted after performing a minor operation in the union hospital of a country town. The typical mulberry-rash was detected upon the wrists, though no other case of the disease had appeared for some months in the district.

The difficulties in diagnosis as a rule disappear after the eruption becomes evident; the dull-red or livid spots, when taken in association with the sub-cuticular mottling, may be regarded as pathognomonic when fully developed.

Measles may be differentiated from typhus occurring in a child, though the eruption of the latter disease may cover the face, by the marked bronchial symptoms, coryza, and nasal catarrh which are obvious in the very early prodromal stage. The measles-eruption is always brighter, more extensive and papular, shows a tendency towards the formation of crescentic groupings, and no swelling of the spleen can be detected. In adults the eruption of typhus is practically never seen upon the face.

Purpura with marked febrile symptoms can only be mistaken for typhus when the spots are small, but the sub-cuticular mottling is absent and hæmorrhages are present from the mucous surfaces.

Meningitis may be closely simulated by typhus, but no rash appears, and the predominance and continuousness of

the intense headache of meningitis is characteristic, since the headache of typhus disappears as soon as marked delirium supervenes. The dull languid heaviness of the expression in typhus is in marked contrast with the irritability and hyper-acuteness of sight and hearing, which cause distressing photophobia and exacerbation of the cephalalgia by loud sounds and vibrations. The pulse is full and bounding, cerebral vomiting is often present, ocular paralysees supervene, and the *tache cérébrale* is well marked; often convulsions occur, and the pupils are frequently irregular.

Hæmorrhagic small-pox, before the appearance of its eruption (which is, moreover, often suppressed), may be mistaken for typhus, but the copiousness of the free hæmorrhages coming from all parts of the body, as well as the extravasations under the skin, taken together with the absence of delirium, leave usually no doubt after the expiration of twenty-four hours. In the ordinary *purpuric* form of small-pox, though free mucous hæmorrhages are usually absent, the skin-petechiæ, vibices, and larger ecchymoses are always more copious than any similar phenomena ever witnessed in typhus, unless when this latter disease is complicated by purpura. The typical shotty papular eruption of ordinary severe small-pox can never be confused with the maculæ of typhus, and the temperature drops in small-pox after the appearance of the exanthem, whilst it remains high in typhus.

Delirium Tremens.—At its onset typhus sometimes in its delirium resembles this affection, but the mental condition is usually markedly different; the mind, wandering from subject to subject, rarely manifests any fixed delusion for more than a few minutes at a time in the disturbance following alcoholism. The tremor is marked from the commencement; visual hallucinations predominate; there is little or no fever at first or all through the attack, the skin being cool and moist; there is never a marked initial rigor, and sleeplessness is a prominent feature from the beginning, and before the mental symptoms appear. Typhus occurring in an alcoholic subject may, however, cause a serious diagnostic problem until the presence of the rash clears up the difficulty.

Pneumonia.—When the apex of the lung is involved the diagnosis of pneumonia from typhus is especially difficult, since often the signs of consolidation are latent, and the disease may be ushered in with delirium or other marked cerebral disturbances resembling the typhoid condition. The appearance of the rash will settle the diagnosis conclusively in all cases of typhus, whilst the physical signs in pneumonia are rarely found absent for more than forty-eight hours if carefully sought for. When pneumonia complicates typhus at an early stage there is seldom a difficulty in the recognition of its secondary nature, since, if the rash is not already apparent,

it will make its appearance in a period rarely exceeding a matter of hours.

Hypostatic pneumonia in typhus, as in all other allied pyrexial states, should not be mistaken for a secondary lobar pneumonia. It is invariably bilateral, rarely affects the whole of both lower lobes, and true pneumonic consolidation as evidenced by tubular breathing is seldom elicited.

Cerebro-spinal meningitis at its onset may be mistaken for typhus, and even after the appearance of an eruption there may be a difficulty, since the rash in this disease, in some of its varied forms, may closely resemble the typhus exanthem. The marked retraction of the neck and the muscular spasms should lead in a case of difficulty to the examination of the cerebro-spinal fluid obtained by lumbar puncture, which will clear up the diagnosis.

Relapsing fever, with its very abrupt onset, high pyrexia, and remarkably sudden crisis, has these features in common with typhus, but the fact of an established epidemic of either disease being present leaves little difficulty. Both diseases are the result of deprivation and destitution, and at the commencement of an epidemic the nature of the fever must remain undecided for some days. Moreover, both diseases sometimes coincide or follow each other in epidemics separated by short intervals.

The course of relapsing fever is totally different from that of typhus, though the onset may be almost identical; the abrupt crisis may appear upon the fourth day, usually it is seldom delayed beyond the end of the first week, immediately after which the patient is able to move about till the relapse occurs seven days later, and the specific spirillum or spirochæte is easily found in the blood obtained during a paroxysm of the fever.

Malaria, in regions where it exists, is differentiated in a similar manner by the recognition of its specific causal organism when a typhus epidemic is also known to be present.

Uræmia is a condition liable to be mistaken for the late stage of typhus in which the typhoid state has become well-marked. Obviously uræmia cannot be confused with typhus if the temperature is normal, as it almost invariably is in the late stages of Bright's disease, where it is most frequently met with in the cirrhotic kidney. But in old septic bladder cases with retention or obstructed flow, where an ascending nephritis or pyelonephritis has supervened, the condition, with its high temperature, may be almost identical with the typhoid state. There will be no eruption, however, in uræmia unless a septic rash or a drug-dermatitis is present, such as might be caused by boric acid or copaiba. The history will almost certainly give a clue to the diagnosis in all such

instances. The writer can recall two cases of *hepatic abscess* sent into hospital as cases of typhus fever during the prevalence of a typhus epidemic, one of which was not differentiated till after death.

Influenza, with its sudden onset, great mental and physical prostration, accompanied by severe cephalalgia and high fever, may closely simulate typhus, especially if an antipyrin or other drug eruption be also present. The short duration of the fever and the intensity of the backache, muscular and joint pains, are usually very distinctive in influenza, before the normal period at which the typhus-rash appears.

Typhoid Fever.—It is only within a comparatively recently period that typhoid or enteric and typhus fever have been demonstrated and recognised as separate and distinct affections. As usually seen, typhus can only be confounded with typhoid fever by those who have not previously seen the characteristic mulberry-rash of the disease. The early appearance, macular character, dirty-red or livid colour of the spots, which come out all together and neither disappear on pressure nor appear in successive crops, and are always more profuse than the raised, isolated, and sparse rose-papules of typhoid, which are never petechial—these features constitute such fundamental differences that a mistake can hardly be made when ordinary care is taken; the sub-cuticular mottling is never evident in typhoid fever. The congested and dull countenance, with suffused conjunctivæ and often contracted pupils and the flat abdomen, contrast markedly with the pale face enlightened by the hectic flush and the bright conjunctivæ and dilated pupils usually seen in typhoid with its distended abdomen. When typhus fever was common in former years the writer often noticed the accuracy with which experienced nurses recognised it on the first admission of the patient to the fever-wards by the characteristic odour given off from the skin.

The temperature often affords little help in the early diagnosis, but Wunderlich's rule may be accepted as proving that the fever is not typhoid if the thermometer registers 104° upon the first or second day of the illness. The presence of epistaxis is strongly presumptive of typhoid. Widal's test is valuable in the differential diagnosis only when it is known that the patient has never suffered from typhoid fever previously, as his blood may give a positive reaction for years after the attack. If this fact be forgotten, a mistake is liable to be made in regarding typhus as typhoid when reliance is placed alone upon the agglutination of the bacilli. Ehrlich's diazo-reaction is useless, as it is as commonly present in typhus as in typhoid. The only absolute solitary positive test of typhoid fever is the identification of the specific bacilli in the blood, stools, or urine, which is now a comparatively

easy matter, even in the earliest stage of the disease, by the newer methods of blood-examination.

Prognosis.—The mortality of typhus, when all ages are included, is probably little over 10 per cent. Hospital-statistics give a death-rate of about double this; but children are frequently treated at home, and the most serious cases as a rule find their way into hospitals. The mortality in early life, between the ages of ten and fifteen years, is only a little over 2 per cent., whilst that of cases between fifty and sixty is over 60 per cent., and at a later age it reaches almost 80 per cent., few very old subjects recovering.

Alcoholism deepens the prognosis at all ages in which it is usually met with, and probably from this cause the death-rate is highest in males. Individuals engaged in prolonged mental work bear the disease badly, and great muscular development and obesity add to the gravity of the outlook. Pregnancy is a less serious item in the prognosis than is the condition following prolonged lactation. Death in the majority of cases occurs from the profound toxæmia influencing the integrity of the myocardium, and this is usually most likely to occur when the temperature has been continuously high with but slight morning remissions or none at all during the second week. As already stated, the gravity of the case is usually proportionate to the amount and livid or petechial type of the eruption. A pin-hole pupil, hiccough, delirium ferox, coma-vigil, a pulse beyond 125 with marked compressibility, and convulsions, should call for a very grave prognosis.

Treatment.—There is no specific treatment for typhus: no known drug or agent exercises a lethal action upon the micro-organism; nevertheless, much may be done to guide the patient safely through his fourteen days of storm and stress. He should be sent at the earliest possible moment to a bed placed in a large well-ventilated and well-lighted room in an upper storey. There is no other diseased condition, save small-pox, which demands such abundant and continually changing amounts of pure fresh air; 3,000 cubic feet of air-space are necessary both in hospital and private houses. This would mean a room of fifteen feet each way, with a thirteen-feet ceiling; for it must be remembered that two individuals at least are always using this air-supply, since the patient cannot safely be left alone for one moment, and when the delirium is active or wild more than two strong nurses will be necessary both day and night.

A woven-wire under a hair mattress and light bed-clothes are desirable. The diet should be liquid, and when the patient can digest milk no other article need be employed, except a weak soup or beef-tea. Abundance of water should be allowed from the beginning; hence the milk may be given

freely diluted with barley-water or any plain aerated water. Often ice is gratefully appreciated, and there is no reason why it should be withheld. The ingestion of abundant fluid materially assists in the elimination of the toxins of the disease.

Cold or tepid sponging should be assiduously employed as a routine in every case where the temperature approaches 103° , and in order to carry this out it is often necessary in the case of adults to have two nurses on duty in the daytime in a severe case of the disease. Chemical antipyretics are entirely contra-indicated on account of the grave condition of the cardiac muscle, quinine only being admissible under certain circumstances. Internal antiseptic agents are useless. The main idea should ever be kept before the mind of the physician and nurses, that if the patient can be kept alive for thirteen days the fever will resolve itself, and with this always in view every exertion upon the part of the patient which entails a demand upon his vital or physical energy must be continuously avoided. Thus struggling with him in his delirium should be minimised to the utmost extent, and to avoid this the nurse should be directed that it is much easier to restrain a patient by keeping him upon his back or side in bed, partly by moral, and if necessary also by physical, force, than to permit him to get out upon his feet, when a serious struggle is sure to follow. Some patients in delirium ferox must be restrained by fastening down the upper sheet.

Alcoholic patients require some stimulant from the commencement, and the necessity for alcohol is decidedly more clearly indicated in typhus than in typhoid fever; but as a rule its administration should never be regarded as a routine, except in the case of the aged, and it is well when possible to avoid it till the disease has entered upon the second week. Children seldom require alcohol in any form throughout the attack. In some cases within sight of the crisis life may be sustained upon stimulants when all else fails; but it must always be remembered that life can be sustained upon alcohol alone for a very short time only. The condition of the heart and pulse is the main indication for the exhibition of this agent and for the regulation of the dose, which as a rule is required in larger quantity than in typhoid. Hence a severe case towards the end of the second week may sometimes demand a daily allowance of 20 oz. whisky or brandy.

Digitalis and strophanthus as agents for the relief or prevention of cardiac failure are not to be relied upon; the former drug may cause serious danger to life by suddenly contracting the arterioles in front of a ventricle whose muscular power has been seriously curtailed or diminished by the rapid degeneration produced by the toxins of the disease. In emergencies the hypodermic injection of strychnine should

be resorted to, and, as pointed out by Sir Lauder Brunton, lime-salts are valuable in cardiac failure arising from any cause.

Complications must be met by agents employed upon approved therapeutic lines. Hyperpyrexia and coma should be met by the use of the cold pack, which is safer and more easily managed than the ordinary cold bath. Severe or persistent insomnia often yields to the ice-cap or cold pack, and active delirium may be reduced by similar means. The newer hypnotics—trional especially—are useful, and morphia may be safely given when the insomnia is not associated with marked delirium and when there is no pneumonia present, or when signs of hypostatic congestion are but trifling. When the latter-mentioned conditions have developed, full doses of paraldehyde are safe and usually effective in combating sleeplessness.

The condition of the bladder requires careful attention, and it is unnecessary to add that every means should be employed to prevent bed-sores.

During convalescence adult patients should be prevented for at least two or three weeks from assuming the horizontal position when the heart remains feeble, and it may be necessary at first to restrain the ravenous appetite which speedily shows itself after the crisis has been safely passed.

URÆMIA

This is the title given to the serious condition of toxæmia which arises when the functions of the renal organs are suspended by organic disease. The old view of the nature of the poisoning is implied in the name 'uræmia,' the condition being considered to be simply the result of the urea circulating in the blood because of its non-elimination. Another theory invented for the explanation of the phenomenon (after the demonstration of the fact that urea failed to produce the symptoms of uræmia) was that the retained urea was converted into carbonate of ammonia, and the resulting toxæmia was consequently regarded by Frerichs as ammonæmia. There cannot be a doubt that various substances, as creatin and allied bodies, play a considerable rôle in the production of the symptoms styled 'uræmia.' The important question still remains to be settled whether the presence of the really unknown poisonous substance is to be regarded as simply due to the faulty elimination of a toxin normally present or to the manufacture in the body of some poisonous substance as the result of an abnormal alteration in the general metabolic processes, as it is contended by some authorities that the toxic substance is different entirely from anything found in

healthy urine. The fact has been placed beyond doubt that the serum of blood taken from a patient in the so-called uræmic condition possesses obvious toxic qualities. Rose Bradford found that removal of over three-quarters of the total kidney-weight in animals was followed by considerable alteration in the general metabolic activity, as seen in the great increase in the production of urea. The remnants of kidney left in the body were found to keep up the excretion for long periods of a much greater total quantity of both urea and water than the average eliminated during health. Hence it becomes evident that the increase of urea and extractives found in the blood and tissues under the above conditions is in no way the result of diminished elimination, but rather points to the probability of the existence of another function of the kidney. In the investigation of this second office of the renal organ probably will be found the explanation of the condition now recognised under the title of 'uræmia.'

Only a theoretical importance, however, need be attached to these hypotheses or to the view that the uræmic symptoms are the result of œdema occurring in the cerebral tissue, since the indications for treatment are firmly established and clear, though the nature and origin of the toxic substance remain unknown. There is little reason to believe that the symptoms are the result of the retention of potash-salts in the blood, as held by some.

Symptoms.—These are varied in their mode of onset, in their nature, degree, and intensity, as well as in their localisation, due to the selective action of the poison for certain organs and different parts of the nervous system. The main symptoms have been already described in the article on 'Bright's Disease,' but a brief *résumé* of them may be here given.

Various types of uræmia have been recognised, as the acute, chronic, convulsive, comatose, respiratory, delirious, delusional, maniacal, amaurotic, paralytic, gastro-intestinal, auditory, &c., but these often pass into each other, and it is common to find many of the symptoms combined at the same time in the same individual. The most chronic type of uræmic symptoms may suddenly take on all the characteristic features of the acute, or the latter may appear without warning in their severest form, and afterwards merge into the clinical picture of the most typically chronic form.

In considering the causes of these marked variations in the intensity and nature of the symptoms, the question arises of the dual or multiple nature of the toxæmia and of the idiosyncrasy of the patient. A more important aspect of the question, however, since it is one of value in the diagnosis, prognosis, and treatment of the condition, is the subject of dosage and of acquired toleration.

Most drugs which act primarily upon the nerve-centres, as best seen in the group of narcotics, tend after repeated doses to induce a changed condition of the cerebral cells, probably of a chemical nature, which within certain limits protects the brain-tissue from their action. Moreover, the repetition of and the intervals between the doses appear to considerably modify the action of such drugs. These facts are obvious in the variations of the action of drugs like opium and alcohol, which in full doses cause profound coma in those unaccustomed to their use, whilst the only symptoms observable in many who become victims to the opium and alcohol habit may be the stimulating effects, often eventuating in permanent excitability, restlessness, and confirmed insomnia. Though the nature of the action of the poison or poisons which cause uræmia is essentially different from that of the typical narcotic, it seems certain that this action is considerably modified by what may be regarded as habit. Thus the sudden production of the poison in the body of a previously healthy individual who is attacked by an acute renal disease usually manifests itself by an explosive convulsion or by a profound coma, whilst a larger dose in the case of a victim of chronic cirrhosis of the kidney may produce no symptoms at all.

This consideration probably throws some light also upon the nature of the toxæmia, which can hardly be alone due to the mere non-elimination of toxic products normally present in the urine, since in one variety of the condition the ordinary symptoms of uræmia may remain entirely *latent*. Thus in the form of anuria, which arises from sudden blocking of both ureters, and which is sometimes witnessed in the total suppression of urine in hysteria, collapse, and certain conditions of profound blood-poisoning, there is often no toxic symptom present throughout, though death may be delayed as long as fourteen days or complete recovery may ensue. (This condition due to obstruction is known as *latent* uræmia.) In these cases it may be assumed, since the question of a slowly acquired toleration is out of the question, that the specific poison is not generated; hence the probability of the correctness of the view that altered metabolism plays a very important rôle in the production of the toxic agent or agents, or that the condition is due, as believed by Brown-Séquard, to some disturbance in the production of an internal secretion supposed to be manufactured by the kidney, such secretion, if it exists, being probably not interfered with in simple anuria.

Of those symptoms which are directly traced to the cerebrum and nervous system, the most common is an intense headache, usually of a most markedly persistent character; it may be confined to the occipital region or extend downwards to the

back of the neck, or be only experienced behind the eyeballs or at the back of the orbit, as in severe migraine, but often it appears to involve the entire cerebral mass. This headache may be the only symptom present, and may pass off completely for a time or altogether in acute cases under the influence of eliminatory treatment. It is, however, frequently the herald of an attack of uræmic convulsions or coma, and is often accompanied by total blindness independent of any retinal changes; sometimes complete deafness may be also observed to be present before the loss of consciousness. Both these latter conditions are transient should the patient survive the attack.

The convulsions may, however, come on abruptly in rare cases without initial headache, blindness, or deafness. They are of the type seen in ordinary epilepsy, and very occasionally the premonitory cry may be heard, though usually it is absent. The face, at first pallid, becomes livid during the attack, and the entire muscular system is convulsed by quickly recurring clonic spasms, preceded by, alternating with, or followed by tonic or tetanoid contractions. Sometimes the contractions are unilateral: they usually tend afterwards to become universal, though the preponderance of the spasms on one side is very commonly marked; and occasionally they may be of the localised typical Jacksonian character. They exhibit all degrees of severity, from the slight muscular twitchings and tremors of the limbs commonly seen in the more chronic forms of uræmia, to the profound general convulsions characteristic of the severest type of epilepsy. As the patient passes out of one fit another succeeds, usually after a very brief interval, during which he is generally quite unconscious. The temperature is generally normal or sub-normal; the tongue may be wounded during the convulsions and a bloody foam appear about the mouth, as in ordinary epileptic seizures; the pupils are usually dilated during the fit and do not respond to light, and nystagmus may be marked. After the cessation of the convulsive movements the patient usually passes into a comatose condition, from which he cannot be aroused; but sometimes he regains consciousness after a variable period of stupor, and often the knee-jerks are absent for a time.

The comatose form of uræmia, though most commonly preceded by a convulsive attack, may set in suddenly without warning in some cases; more usually, however, severe headache precedes it, and muscular twitchings of the face or hands generally may be observed, though these sometimes are so trivial as to escape notice. In a case of uræmic coma only preceded by headache, or even where this has been absent, convulsive seizures may follow later on, though sometimes death results without the supervention of any spasms.

In cases of uræmic convulsions and uræmic coma occurring in acute Bright's disease, the patient may recover completely, though temporary blindness, deafness, transient aphasia with or without hemiplegia, or any form of monoplegia or anæsthesia may be also present. The fits of both coma and convulsions arising during the progress of a progressively hopeless chronic renal affection may also be recovered from under active therapeutic treatment, though their return at a later stage is certain unless the patient's life be terminated by a cerebral hæmorrhage or other complication. In these chronic cases the coma may never be absolutely complete, a condition of somnolency being maintained for many days, during which the patient may be more or less completely aroused, to pass again into a state of torpor in which muscular twitchings are commonly observed from time to time. Hiccough and Cheyne-Stokes respiration are common under these latter conditions.

Sometimes, though rarely, the cerebral manifestations of uræmia show themselves in some obvious form of psychosis, as acute mania, melancholia, or other form of delusional insanity; occasionally the mental condition resembles the fitful delirium met with in pyrexial and other diseased conditions.

Respiratory symptoms sometimes dominate the clinical picture and mask the underlying toxic condition. They manifest themselves usually in a form of dyspnœa which commonly occurs at night in paroxysms resembling asthma. Sometimes the difficulty of breathing is continuous, though exacerbations occur and Cheyne-Stokes respiration may be marked. The breathing may also be accompanied by hissing noises, stridor, or other symptoms suggestive of laryngeal or tracheal origin; insomnia may be marked, though there is no headache or twitchings pointing to the nature of the respiratory disturbance.

The uræmic symptoms arising from disturbance of the gastro-intestinal tract may be either peripheral or centric. The latter resembles the vomiting so constantly met with in cerebral affections, and may be uncontrollable, no other evidence of uræmia save the albuminous urine being present. Another type of vomiting commonly met with in chronic cases has all the features of a gastritis. It is probably due to the elimination of the retained products in the form of ammonia, since the ejecta and breath have a strong ammoniacal odour and alkaline reaction, and the symptom may be effectually checked by the administration of lactic or other organic acid.

Diarrhœa may occur independently of vomiting, and it likewise appears to be due to the peripheral irritation caused by the decomposition of the urea-like products. The mouth

suffers in a similar manner, and a form of stomatitis is recognised as due to the uræmic condition in which there is considerable glossitis and erythematous redness of the mucous membrane, involving also the pharynx, so that, in addition to salivation, pain may be experienced in both chewing and swallowing.

The more chronic and minor symptoms of uræmia really only differ in degree from the more urgent manifestations. Thus headache of a dull continuous kind is common, drowsiness, or a lethargic stupid mental condition which may never end in unconsciousness, persistent nausea, temporary dimness of sight, deafness, or tinnitus—these may come and go sometimes with muscular twitchings, but as a rule when these latter appear a convulsive or comatose seizure is not far off. There is often in the more chronic cases a considerable degree of itching of the skin, which is usually dry; and not uncommonly various erythematous and urticarial or eczematous rashes occur, which are probably due to efforts on the part of the skin-glands at the vicarious elimination of irritating retained products. A urinous or ammoniacal odour may not only be perceived from the breath, but also from the skin, and occasionally the urinary salts have been observed to remain as a crystalline powder upon the cutaneous surface after the drying up of a concentrated but scanty vicarious sweating (*uridrosis*). The irritability of the muscles may manifest itself in severe cramps involving the calf of the leg or other parts.

Fever is often absent throughout; but in the last stages of the uræmic state there is a tendency to inflammation, especially of the serous membranes, and the resulting pleuritis, peritonitis, or pericarditis may be accompanied by a high pyrexia, though a purulent secretion of considerable amount may be found in the peritoneum with a normal or sub-normal temperature. Occasionally a series of severe uræmic convulsions may be observed, in which a high temperature is registered without any inflammatory lesion to account for the pyrexia. The typhoid condition which supervenes in diseases of the urinary tract originating in bladder-mischief from obstructed flow is caused by a combination of the symptoms of uræmia and of those due to other toxins.

Diagnosis.—The chief point in the diagnosis obviously lies in the examination of the urine, which is so constantly found to be highly albuminous in all acute cases. Practically the diagnostic problem resolves itself into the recognition of the form of Bright's disease present. The amount of urea found present in the urine is generally little, if any, criterion of the probable advent of the condition; and this is easily explained by the consideration previously referred to, since a small dose of the poison in an acute case is more likely to

produce serious symptoms than a much larger dose in an organism long habituated to the toxin. Moreover, the poison in the blood, whatever may be its composition, does not necessarily bear any proportion to the poverty of the urine in urea-compounds. It is not uncommon, for example, to observe instances of chronic cirrhosis of the kidney in which the s.g. of the urine approaches the density of distilled water, without the presence of any toxic symptom, though vascular and cardiac changes are present in an advanced degree; whilst, upon the other hand, the urine in an acute post-febrile nephritis may show little diminution in the amount of urea excreted, though the patient is suffering from uræmic convulsions or coma. It is in the former condition of affairs that a mistake is liable to be made when toxæmia does occur, because there is often no anasarca, and the amount of albumin in the urine may escape detection owing to its smallness.

Brain disease.—The condition of the heart and arteries and the state of the blood-pressure are of great importance; but these points do not really assist in the elimination of *cerebral hæmorrhage*, since this complication is a common terminal event in chronic Bright's disease. The exclusion of organic cerebral lesion is therefore a matter of great difficulty and sometimes of impossibility. When hemiplegic signs, conjugate deviation of the eyes, and ocular or other form of paralysis are present, the probability of a cerebral apoplexy is greatly increased. The form of brain-lesion most likely to be mistaken for uræmic coma is a hæmorrhage into the pons, since in this latter condition, if the clot be central, there will be profound coma, pin-hole pupils, and double hemiplegia, all four limbs being involved, convulsions being also not uncommon, and sometimes (more frequently than in uræmia) high temperature is present.

Conjugate deviation—the condition in which both eyeballs are rotated to one side in cerebral lesions—should be looked for; in ordinary hemiplegia the eyes look towards the site of the lesion in the brain, but when convulsions occur in the paralysed limbs the eyes look away from the cerebral lesion towards the convulsed side. In hæmorrhages into the upper part of the pons ordinary hemiplegic symptoms follow: when the lower portion is involved crossed hemiplegia results, and the eyes look away from the site of the brain-lesion. The demonstration of any of these points is almost conclusive of the diagnosis of cerebral hæmorrhage; but Osler has recorded cases where the *post-mortem* revealed an ordinary hemiplegia to be the result of the uræmia, no lesion being visible in the brain. The lightning-like suddenness of a cerebral hæmorrhage is in marked contrast to the slower onset of uræmia, but this is not always the case.

Puerperal convulsions, or puerperal eclampsia, may be regarded as a manifestation of uræmia, but the condition often differs from the typical uræmic form by the absence of marked coma. This is probably, as suggested by Gowers, owing to the powerful cause of the reflex irritation present in the uterus in the pregnant condition, which of itself involves an abnormal state of the nerve-centres, as shown by the occurrence of such functional disturbances as tetany and chorea. The view of the microbic infection of the blood in puerperal eclampsia need not be considered in this connection, as the highly albuminous condition of the urine almost invariably present shows that the renal function is in abeyance. It is worth remembering that in this variety of uræmia a very high temperature is often recorded.

Diabetic coma resembles the uræmic variety closely when this exists without muscular twitchings or convulsions and has been preceded by dyspnœa. The main diagnostic point is the detection of sugar in the urine and the characteristic apple-like odour of the breath where the history of the case does not demonstrate the diabetic nature of the attack.

Opium-poisoning is differentiated by the pin-hole pupils and the slowness of the respirations, accompanied by a slow and full pulse, and frequently by the characteristic odour in the breath of the crude drug or of its tincture; an important point also is that the extreme contraction of the pupils is always bilateral and equal. The face is cyanosed, and the skin cool and moist.

Alcoholic coma is recognised by the odour of the spirit in the breath; the pupils are usually dilated as in uræmia, and are equal; the respirations are deep and often stertorous, and considerable lividity of the face occurs. A very valuable sign may be demonstrated by the thermometer, which in all severe cases shows that a marked drop in the body-temperature exists when the coma is profound. The same test applies with even greater force in *chloral-poisoning*. The usual accompaniments of uræmia, as twitchings, convulsions, albuminuria, anasarca, retinal changes, and cardio-vascular lesions, are commonly absent in these as in most other forms of coma occurring independently of renal disease.

Epileptic convulsions with *coma* closely simulate uræmia, as already incidentally mentioned. The history of the case seldom leaves a doubt; the aura and initial cry followed by the convulsion, during which urine is often passed, and which lasts usually but a few minutes, after which the cyanosed patient falls into a deep sleep, are symptoms whose history is usually obtainable from the friends of the patient. In cases of doubt the examination of the urine is conclusive, save in those rare instances where albumin is found in it.

Hysterical convulsions and *coma* are less liable to be mistaken for uræmia, the preceding psychic excitement being highly characteristic. The convulsive movements are rarely rhythmical, and they show more or less purpose, and may be of much longer duration; the tongue is never bitten, rambling talking or screaming may continue throughout the seizure, and the subsequent coma is never profound. The attack at any stage may usually be brought to an abrupt termination by a dash of cold water.

The convulsive seizures occurring during *strychnine-poisoning*, *tetanus*, and *hydrophobia* can only be mistaken for some form of puerperal uræmia, since consciousness remains intact throughout; the seizures which occur in *tetany* may simulate the twitchings of an approaching uræmic convulsion, but the limitation of their distribution to the hands and feet, and the characteristic position assumed by the fingers, are diagnostic.

Sunstroke-coma bears some resemblance to the uræmic form, the unconsciousness being usually profound, and convulsions and suppression of urine often occur in the hyperpyrexial form. The diagnosis is, however, easy, the history of exposure to extreme heat or violent muscular exertion, or both combined, is always forthcoming, and the body-temperature is excessive.

Prognosis.—The outlook in uræmia is that of the form of primary renal affection of which it is the direct outcome. In the acute form of Bright's disease and in puerperal eclampsia the prognosis is hopeful, when prompt treatment can be initiated. Even in the most unpromising chronic cases a fatal issue may be averted for a long period.

Treatment.—This has been dealt with in considering the management of Bright's disease. The main reliance must be placed upon eliminatory methods. The condition of the kidney as a rule forbids the exhibition of diuretic agents. Vicarious elimination must therefore be effected by the skin and intestinal mucous membrane. By the administration of saline cathartics the specific poison and its allied urea-like substances may be eliminated in such quantities as may remove the symptoms completely. Powerful diaphoretics, as the hot-blanket bath, warm pack, air-bath, &c., supplemented by pilocarpine hypodermically *after the skin begins to act*, may be alone available when the patient is unable to swallow. Where the convulsions and coma fail to yield to the action of these agents there should be no hesitation in attacking the blood itself. Thus, in an apparently hopeless case, where the convulsions had lasted for a week, the writer had venesection performed to the amount of about 40 oz., after which several pints of warm normal saline solution were injected into the loose cellular tissue in several

regions of the body, and a speedy and permanent recovery followed the relief given to the acute renal disease.

The symptoms may demand special treatment; insomnia, headache, delirium, and even the convulsive seizure itself may be relieved by chloral or by chloroform-inhalation. Morphia is a drug to be used with the greatest caution, or not at all in the majority of cases. Lumbar puncture, according to Rose Bradford, is capable of producing temporary relief from both the coma and severe headache. Vaso-dilators—nitrites, erythrol, nitroglycerin, &c.—are useful to diminish the increased tension accompanying the condition, and the authority just mentioned does not hesitate to employ digitalis and caffeine when the blood-pressure is low after the heart dilates.

URTICARIA, or NETTLE-RASH

This is the name given to a cutaneous affection in which wheal-formation characterises the eruption.

Etiology.—The condition can be always produced in healthy individuals by the application to the skin of the common stinging-nettle, and hence this has given origin to the popular name of the affection.

Other external irritants appear to require some predisposing condition to be present, since they fail to produce the wheals in most individuals, whilst they may be always potent in others. This idiosyncrasy is often demonstrated by the bites of fleas, mosquitoes, gnats, bugs, and other parasites, the stings of wasps, bees, and jelly-fishes, and contact with certain hairy or woolly caterpillars, &c.

In individuals strongly predisposed to such external irritants their susceptibility may be sometimes demonstrated by the wheal-formation which follows simple mechanical stimuli applied to the skin. Thus, the so-called *factitious urticaria*, a condition allied to *dermographism*, may be produced by stroking the skin with a sharp pencil, and sometimes the letters of a word traced in this manner upon the cuticle may distinctly appear in a raised form in a few seconds.

Internal irritants are the main factors in urticaria in the great majority of cases, whether the patient be predisposed or not. Amongst these the most common are shell-fish, crabs, lobsters, mussels, tinned meats, pork, various vegetable substances, as mushrooms, strawberries, apples, &c. Poisons manufactured in the intestinal canal during altered conditions of the digestive process sometimes produce urticaria, and the affection has been known to arise from the absorbed products or secretions of parasites living in the bowel, or lodged in the tissues as in the case of hydatids.

Many drugs give rise to urticarial eruptions in some susceptible people, as cubebs, copaiba, quinine, iodides, salicylates, santonin, &c.

Abnormal blood-conditions arising in such diseases as gout, jaundice, asthma, malaria, and uræmia, and in the pregnant state, hyperlactation or ovarian irritation in susceptible individuals may also induce the cutaneous affection. It is often observed after the injection of the various anti-toxic-serum preparations, and was frequently observed before blood-transfusion gave place to the injection of normal saline solution.

Pathogeny.—The generally accepted explanation of the wheal-formation is that it is a vaso-motor phenomenon produced by alterations in the lumen of the vessels, caused by the reflex irritation of sensory or vasal nerves, allied to the condition recognised as angio-neurotic œdema. Paralysis of the vaso-constrictors or irritation of the vaso-dilators may be present, the circumscribed œdema of the skin accounting for the production of the wheal, which is white in the centre through subsequent pressure on the vessels caused by the exuded lymph. Other factors are supposed to be contraction of the muscular fibres of the skin, and spasm in the venous coats which retards the absorption of the exuded fluid.

No theory of the production of urticaria, however, can be accepted which omits the state of the coagulability of the blood, the importance of which was first pointed out by Wright. Paramore has recently tested the coagulability of the blood in a series of cases, and always found it abnormally low. The condition was produced at will by reducing the coagulability, and was always removed by increasing it through the administration of a calcium salt. Of more practical moment was the fact, observed also by many others, that by the use of the latter drug the spontaneous cases were often rapidly cured. The authority just mentioned considers that the excretory power of the kidneys plays an important rôle in diminishing the clotting power of the blood; when the renal function is in abeyance the salts retained in the blood prevent or retard the formation of a coagulum. (See page 1595.)

Symptoms.—Urticaria appears as a rule suddenly. The wheal, which is the characteristic lesion of the disease, shows itself as a raised patch of skin, which may measure as little as a lentil or as much as the palm of the hand; large and small ones may be seen in the same region, and their distribution has no symmetrical feature. At first the raised patch is usually red, but soon it becomes of almost ivory whiteness in the centre, the margins remaining bright red, and often a narrow zone of redness extends into the neighbouring skin. The duration of the wheal is usually only a matter of hours, but in rare cases it may be indefinitely prolonged. As it

fades away the white central part may often be seen to become red, and the red margins white, but no trace is left of its transient existence after a few hours, and often within a much shorter period. Intense pruritus and burning, tingling, or stinging sensations accompany the appearance of the eruption, so that scratching, which gives a very transient relief, is always freely indulged in, and the irritation of the finger-nails on the skin in the neighbourhood of the wheal brings out immediately numbers of smaller ones, which may coalesce with the original, and in this way patches of œdematous skin may be suddenly produced, or a large continuous sheet be involved. In severe cases the wheals come out in successive crops for several days, and in the *chronic* variety of the condition these may continue to appear for many months or even years, though the life of each elevated patch is usually as brief as in the most fleeting form of the disease.

Any portion of the skin may be affected, and upon the unaffected parts, when the attack is on, the wheals can readily be produced with the slightest local irritation. The mucous membrane of the nose, mouth, tongue, pharynx, vulva, bronchi, and stomach may be also involved in a similar manner.

Many varieties of urticaria have received fanciful names from the shape, coalescence, and distribution of the wheals, as *U. gyrata*, *U. annulata*, &c. Sometimes they remain small (*U. papulosa*), and this variety is practically identical with lichen urticatus or lichen strophulus: a red papule capped by a small dark scab, caused by scratching, is left remaining after the disappearance of the small wheal (see page 1450). A *hæmorrhagic* and a *bullous* variety have been met with, in which bleeding or vesiculation takes place in the swollen patch. It is not uncommon to see considerable traumatic lesions, with bleeding, produced by the violence of the scratching; these may cause pigmentation or desquamation, neither of which phenomena belongs strictly to the urticarial lesion. *Urticaria pigmentosa* is a distinct affection. A special form has been designated *U. œdematosa*, which is met with when the wheals are situated where the skin is lax, as about the eyelids and scrotum. When very large tracts of the skin are involved the wheals may constitute great hard or brawny œdematous swellings, causing remarkable fugitive deformity of the face or other regions; this form is termed *Urticaria gigas*, and seems to be practically identical with *angio-neurotic* or *acute lymphatic œdema*. Its urticarial nature may be demonstrated by the spontaneous presence or by the induction of typical smaller wheals by scratching the skin in an unaffected region. In these giant and œdematous forms there is sometimes little or no itching. As a rule there is no fever, and little if any constitutional disturbance, but in the severe forms, which occur in combination with or as the

result of serious toxæmia of different kinds, the primary condition may be fatal, as in mussel-poisoning and uræmia. When the wheals invade the bronchial surface, asthmatic symptoms may suddenly arise.

Diagnosis.—The sudden appearance, fugitive nature, and resolving character of the wheals, accompanied by itching, and unattended usually with any constitutional disturbance, leave little difficulty in recognising the condition at a glance. The majority of patients already have the diagnosis settled from their previous acquaintance with nettle-stings.

In its giant or massive form the disease may be mistaken for an erysipelatous attack, but the discovery or production of a typical wheal in another part of the skin settles the diagnosis, as it does also when the mucous membranes are involved.

Pemphigus is simulated in the rare cases where bullæ form in the wheals, but the fugitive nature of the eruption is quite characteristic. *Erythema nodosum* is easily differentiated by its characteristic distribution on the shins and elbows, and by its longer duration.

Chronic urticaria may suggest *scabies*, especially in those cases where the wheals only come out after the patient has got warm in bed, the lesions caused by the scratching remaining during the day. The papulose form presents the same diagnostic problem, and *scabies* can only be excluded by the distribution of the lesions and by the demonstration of the absence of the itch-insect and its pathognomonic burrows.

Prognosis.—This, even in the most alarming types of the giant or massive form of the affection, is always good, the only reservation being in those cases where a serious blood-poisoning underlies the condition. The urgent symptoms caused by the invasion of the glossal, pharyngeal, or bronchial surface are usually unaccompanied by danger.

In chronic urticaria the prognosis as to duration should be guarded, as this form of the disease proves often rebellious to treatment of all kinds.

Treatment.—The first element in the treatment in all cases lies in the discovery of the specific cause, the disease rapidly disappearing after the elimination of the toxic agent. In the chronic cases this is often a matter of serious difficulty, and one food after another must be suspected and forbidden till its innocence is established and the offender is discovered. When the abnormal condition is due to intestinal fermentation or gastric indigestion, to the presence of uric acid or gouty conditions, the obvious indication is to remove the primary abnormal state by suitable agents, as purgatives, salicylates, alkalies, and digestive agents. In all cases the urgency of the symptoms may be relieved, and in many entirely removed by Wright's plan of rapidly increasing the coagulability of the

blood by the administration of calcium chloride in full doses. The itching, when it is due to pressure of the blood and lymph in the capillaries in the wheal, as is supposed to be the case by Meachen, may be relieved by local applications consisting of carbolic acid or other local anæsthetic; but as it is more frequently due to the presence of the toxin circulating in the blood, eliminatory measures must be trusted to.

URTICARIA PIGMENTOSA

This is a rare disease, appearing almost invariably in early infancy, characterised by the presence of urticarial manifestations which, instead of resolving, remain for an indefinite time more or less deeply pigmented in the form of raised or flattened nodules or maculæ.

Etiology and Pathology.—There are important reasons for considering this diseased condition as a separate entity entirely distinct from ordinary urticaria, though the presence of factitious urticaria can usually be demonstrated by the ease with which wheals and dermographic phenomena can be produced when the skin is irritated mechanically in the vicinity of the nodules. Digestive troubles have nothing to do with the production of the condition, and recent researches have established the fact that there is no deficiency of calcium salts in the blood; the pruritus which is such a prominent feature in ordinary urticaria is sometimes entirely absent or only exists to a trifling extent.

The appearance of the disease shortly after birth is indicative of some congenital developmental error, and Graham Little defines this as a congenital tendency to the over-production of *mast-cells*, somewhat analogous to the developmental errors causing hæmophilia and other conditions, but the affection is not a family-disease. These cells are large, closely packed, and hence cuboidal in shape, with coarse basophile granules, and are arranged in columns round the blood-vessels and sweat-ducts, separated by wide lymph-spaces, caused by the œdematous effusion into the papillæ. The brown coloration is the result of the accumulation of pigment derived from the hæmatin.

Symptoms.—The eruption appears suddenly, like urticaria, but apparently with less itching; typical wheals form, and others can be easily produced by scratching or other mechanical irritation of the skin in the vicinity of the initial blotches. These become flattened in time, and may finally assume the form of pigmented macules, the hue of the wheal passing from an erythematous redness to a dull brown, yellow, or buff.

Male children are much more frequently affected than females ; in contrast, it is said, to the opposite condition in ordinary urticaria. The nodules are liable, when in the quiescent state, to exacerbations of activity upon the application of mechanical irritation, so that turgid wheals reappear from time to time. An interesting fact observed by Colcott Fox is that the factitious urticaria produced by irritating the uninvolved skin disappears without leaving pigmentation. Any portion of the body may be affected ; most frequently the eruption first appears about the abdomen, from which it generally tends to invade the limbs ; sometimes the mucous membrane of the mouth shows the characteristic pigmented raised patches, and the soles, palms, and face may be affected. The pigmented nodules and maculæ remain for many years, but gradually disappear, usually before puberty.

Diagnosis.—The diagnosis is usually easy when the early infancy period at which the eruption first appeared is known and its characteristic features recognised. An important character which distinguishes it from all other pigmented lesions is the tendency shown by the remnants of the wheals to become active, raised, and turgid upon irritation, and by the ease with which factitious urticaria can be produced in the neighbouring regions of the skin. The ultimate prognosis is good, the condition being practically certain to fade away and leave no traces of its existence before adult life is reached ; but treatment, except for the relief of itching, should this be present, is useless. Calcium salts have no effect upon the affection.

VACCINIA (COW-POX)

The specific eruptive disease which occurs spontaneously in the cow, and is recognised by the name 'cow-pox,' is sometimes accidentally transferred to man by the virus from the ruptured vesicles situated upon the udders and teats of the affected bovines finding its way into some abrasion upon the fingers or hands of the milker. The immunity afforded by this casual attack of cow-pox against the contagion of small-pox was turned to practical account by Jenner (see page 1602), though the existence of such protection had been recognised by previous observers. Human vaccinia is extremely rare, save when it is intentionally produced by the inoculation of the fluid contents of a vesicle of cow-pox in the human subject by the operation known as *Vaccination*.

The literature of cow-pox and vaccination has grown to be voluminous, and many interesting problems still await solution ; thus, though the disease is beyond doubt due to a specific micro-organism, this has never yet been satisfactorily

demonstrated, though many scores of different microbes have been isolated from the contents of the vesicles in man and in the cow. It has been demonstrated that the liquid portion of the vesicular contents, when mechanically freed from the organised ingredients, is sterile.

It may be accepted, however, as practically proven, notwithstanding the Lyons experiments, that cow-pox is 'small-pox in the cow'; thus the vesicles resulting in the calf, from the inoculation of the virus of typical human small-pox, after one or two removes have been made to yield a vaccine, which has been passed through thousands of human beings, behaving in each case in the same manner as does the vaccine obtained from spontaneous cow-pox.

In the horse the disease known as 'grease' is recognised as 'equine small-pox' or horse-pox, and the lymph or vaccine obtained from the vesicles affords an apparently equal degree of immunity against variola when inoculated into the human subject. In the early days of vaccination this equine vaccine was extensively employed, and, according to Copeman, constituted the lymph which De Carro sent out to India.

VACCINATION

When a small quantity of the lymph from a cow-pock vesicle is rubbed into the human arm, after punctures or linear cross-hatching scarification, performed under strict antiseptic precautions, the slight transient traumatic irritation subsides after some minutes or hours. The site remains, with the exception of the presence of the lancet-marks, quite normal till the evening of the third or the fourth day, when a small red papule becomes visible. This by about the fifth day begins to show vesiculation, with a very faint suspicion of an areola of surrounding erythematous redness bordering it. The vesicle by the seventh day is fully formed and plump, its areola is more distinct, it is depressed in the centre, with thickened or swollen edges, which are well-defined, and the clear or milky contents can only be extracted by a number of distinct needle-punctures, showing that the vesicle is distinctly loculated. By the next two days the areola is well developed, and may form a halo of deep redness, extending for a couple of inches; upon the tenth day the vesicle has greatly increased in size and has commenced to pustulate and dry up in the centre, where a crust forms which increases peripherally till the entire site of the inoculation-lesion is covered by a dark-brown scab or crust which usually falls off by the middle of the third week, leaving a livid scar, which eventually becomes white and pitted. Where several discrete scarified areas have been inoculated with the lymph or vaccine, these may, if situated too close to each other, become confluent and covered over with one very large brown scab

or crust. To efficiently protect against small-pox three or four distinct areas should be inoculated, as statistics conclusively prove that the degree of immunity is directly proportionate to the number of distinct vaccination-scars observed upon the arm. The dread of the trifling inconvenience and scarring often tempts physicians to remain satisfied with one or two vesicles, which practice has much to say to the discredit attached to the results when scrutinised by prejudiced critics after small-pox epidemics.

There is some marked but usually trifling pyrexia and mild constitutional disturbance arising about the fourth day, and enlargement with tenderness of the neighbouring lymphatic glands is commonly experienced in susceptible individuals. Leucocytosis is a constant feature about the third or fourth day. Supernumerary pocks or vesicles sometimes form in the vicinity of the site inoculated, and a generalised vaccinia has been very occasionally observed, in which a series of vesicles coming out in successive crops have covered a considerable area of the skin by the seventh day, in marked contrast to the vesicles which formerly appeared about the twelfth day when the small-pox virus was employed for inoculation. More frequently generalised vaccinia has been observed to follow auto-inoculation by scratching the pock and sowing the germs in other regions, as the lips, eyes, face, or hands, by means of the infected finger-nails.

Eruptions of various kinds, erythematous, roseolar, urticarial, eczematous, &c., are not uncommon, and sometimes erysipelas, gangrene, and septic rashes undoubtedly occur from contamination of the lymph; the importance and frequency of these have been grossly exaggerated by unscrupulous opponents of vaccination. The number of deaths annually attributed to vaccination Acland places at about one-seventh the mortality from chloroform-anæsthesia. Of the diseases supposed to be transmitted by vaccination, syphilis is the most important; that such an event is possible cannot be denied, but the risk has been proved by carefully compiled statistics to be so infinitesimal that it may be ignored when ordinary precautions are taken. The German statistics, including 12,250,000 cases of vaccination, do not show a single instance of vaccinal syphilis. The possibility of leprosy and tubercle being inoculated along with vaccinia rests upon such a slender basis that many investigators who have minutely studied the question deny it entirely.

The duration of the protection or immunity conferred by vaccination has been already referred to in the article on small-pox. It may be accepted as a period of many years, but re-vaccination should be performed in all cases after the lapse of seven or ten years from the primary operation, in the presence of an epidemic of the disease. The almost con-

stantly observed modification which occurs in the disease when this attacks individuals in whom the vaccination might be expected to have faded, after the lapse of very many years, need not be again referred to. The important fact is mentioned by Osler that from 1874 till the last report in 1902 no death from small-pox has occurred in the huge army of Germany, save one isolated case under peculiar circumstances, nearly a quarter of a century ago.

VARICELLA, or CHICKEN-POX

This is a specific infectious disease, characterised by a vesicular eruption, coming out in successive crops, usually observed in early life, and generally associated with but trifling constitutional disturbances.

Etiology. —The disease is commonly seen in children about the fourth year of age, the great majority of all cases occurring between the second and seventh years, though instances of it are occasionally met with in the advanced years of life, when the individual has chanced to escape an attack in childhood, as one seizure usually protects or confers life-long immunity.

It often appears in epidemic form, especially following outbreaks of scarlatina, measles, or small-pox; sporadic cases are, however, common in which infection or contagion from contact with other cases cannot be traced. The contagium appears to be readily carried in the air by fomites, and the virus adheres to clothing and lingers for a short period about rooms, and it is believed that it finds its way into the body, through the lungs, in the inspired air. The disease is entirely distinct from small-pox, and an attack confers no immunity from small-pox nor does vaccination afford any protection against chicken-pox. The latter disease cannot be inoculated, and the specific germ has not yet been isolated.

Symptoms. —After an *incubation* period of about the same duration as that witnessed in variola—*i.e.* about thirteen days, which in rare cases may be lengthened by four or five days—the invasion occurs. The first sign of the invasion noticeable is generally the appearance of the eruption, since in children especially the initial fever is so slight as to escape detection before the vesicles appear. The pyrexia is therefore usually first observed in association with the rash. It is rarely severe, except in the case of an adult, where a smart chill, headache, and marked fever may usher in the attack, which is liable to be regarded as one of small-pox. When the initial fever is detected, either in the child or adult, the eruption will follow within twenty-four hours.

The Eruption.—This at first consists of numbers of bright-red spots, usually appearing upon the chest or abdomen, slightly elevated, and hence papular; they disappear upon pressure, but are never shotty. In a few hours the papules are found to have undergone vesiculation; the vesicles steadily increase in size up to the third or fourth day, when they usually attain to the size of a split-pea. They are oval or hemispherical and plump, show no umbilication as a rule, and collapse entirely when punctured by a needle, so that they are not loculated as are the small-pox vesicles. They contain a clear, glistening, colourless fluid, and suggest the resemblance to minute bullæ caused by a sprinkling of boiling water upon the skin. Usually there is no red zone surrounding the vesicle at this stage, though later a faint pink areola is often observable.

After about twenty-four or thirty-six hours the vesicular contents become opalescent and straw-coloured, but true pustulation only proceeds in a few, or does not occur at all, the vesicle bursting or shrivelling up by about the fourth day, when its site is marked by a small dry scab, which falls off and leaves no scar or pit unless when suppuration has occurred, probably the result of scratching or friction. A marked feature of the eruption is its appearance in successive crops, which gradually extend from the trunk to the limbs, invading the face, scalp, and mucous membrane of the cheeks, tongue, palate, pharynx, and sometimes the larynx, conjunctiva, and genitals. Successive crops may continue to appear, sometimes for ten days; hence in a given case at any time the eruption is seen to consist of soft papules, clear and opalescent vesicles, and dry scabs or small crusts. The vesicles very rarely show a tendency to become confluent in any region, remaining as a rule unmistakably discrete and isolated, unless when scratched or irritated, though two markedly confluent cases have recently been reported by Neech and Rolleston. All through the above stages there may be no evidences of constitutional disturbance, and the secondary fever of small-pox is never seen; but when the eruption is very copious, especially in adults, more or less fever may continue throughout, with slight evening exacerbations. In a very small percentage of cases the true vesicular eruption is observed to be preceded by a diffuse scarlatiniform rash, which disappears after the vesicles come out.

Complications and sequelæ are very rare; bronchial or laryngeal irritation occasionally proves troublesome, and has been known in rare instances to end fatally. Nephritis is very occasionally observed, and sometimes sloughing of the cellular tissue. A death from the hæmorrhagic type of the disease, supervening upon the fourth day, has been recently reported by Rundle in the City Hospital, Liverpool.

There are three rare forms or types of the eruption recognised, the first being the *hæmorrhagic*, just referred to, in which skin-petechiæ are associated with hæmorrhages from the mucous surfaces. A *bullous* type of eruption has been sometimes observed which resembles acute pemphigus, the vesicles forming considerable blebs, prone to pustulation through the action of cocci of various kinds.

Varicella gangrenosa is the name given by Hutchinson to the third rare type of the disease. In this, as implied by its name, there is a marked tendency to a gangrenous action setting in around the vesicles, and as the scabs fall off deep ulcers, extending downwards to the muscles, may be observed. This form is usually associated with conditions of neglect and ill-feeding, and has been met with in the subjects of acute phthisis and is always a grave disorder.

Diagnosis.—The only problem in diagnosis is the differentiation of the affection from Variola, and this is not rarely attended by great difficulty. The differential diagnosis is already fully detailed under 'Small-pox,' on page 1614.

The prognosis, as already stated, is almost invariably favourable; the very rare exceptions have been mentioned: indeed, the disease is so very mild that the great majority of cases require no treatment from beginning to end. It is advisable, however, to take precautions against scratching and injuring the vesicles, otherwise considerable pitting may supervene, especially upon the face.

VERTIGO

This, also known as Giddiness, Dizziness, or 'Swimming,' is the term often loosely used to designate:

(1) The objective sign manifested by rotation, turning, or other movement of the body, generally accompanied by unsteadiness of gait, sometimes by actual falling, and usually by more or less transient mental confusion;

(2) The subjective condition experienced by the patient in which there is a sensation of rotation or other body-movement, without the occurrence of any actual motion;

(3) The sensation of the patient in which surrounding objects seem to be in motion, though he is usually conscious that the phenomenon is a deceptive one.

The terms *subjective* and *objective* are often employed in a totally different sense from the above; thus, subjective vertigo is defined as that variety of the disease in which the patient feels as if his body were in motion, in contradistinction to the type of vertigo in which the individual has the illusory sensation that surrounding objects are in motion, this latter variety or type being styled 'objective vertigo.'

Etiology and Pathology.—Vertigo must be regarded merely as a symptom common to many cerebral or other conditions. Thus, it is not unusual to find it well marked in organic brain-disease, especially in tumours involving the cerebellum or pons. In its most severe degree it is met with when the middle peduncle of the cerebellum is affected, in which case the condition is usually not merely a sensation, but actual rotation of the body is frequently observed. It is a very common symptom in the senile forms of arterial degeneration, and may be the forerunner of apoplectic or hemiplegic attacks.

In purely functional cerebral affections, as in migraine and true epilepsy, vertigo is a common symptom. In the latter disease it may appear in its most typical form before the advent of the general convulsion, constituting one of the varieties of aura seen in this affection. The so-called *epileptoid vertigo* must not, however, be confused with the variety of the affection constituting the aura of a convulsive seizure. The term should be strictly confined to the severe recurring type of vertigo which is probably caused by labyrinthine disturbances, and which has no pathological or clinical association with true epilepsy. The name has originated in the abrupt and startling suddenness with which the paroxysm of vertigo is usually ushered in. The writer has seen it in its most severe and typical form in ataxic patients, but it may occur in otherwise healthy young adults.

Ocular vertigo is that variety of giddiness caused by paralysis of one or more of the eye-muscles, producing erroneous projection of the visual field. It is also not uncommon in nystagmus, and is sometimes due to errors in refraction and diseases of the eye.

Various swinging or rotatory movements of the body, as, for example, the motion produced by the rolling or pitching of a vessel at sea, may be the cause of a severe attack of vertigo, as experienced before or during an attack of seasickness.

Gastric vertigo is the name given to the variety of giddiness supposed to be due to disturbance arising in the stomach from an arrest in the digestive process, or from prolonged fasting. It is probable that the majority of such examples are really instances of aural origin, in which the gastric irritation acts merely as the exciting cause of the paroxysm; and the same remark applies to the so-called *cardiac* or *cardio-vascular* variety met with in valvular disease and arterial sclerosis. Probably this is the correct explanation also of the so-called *laryngeal vertigo*, which sometimes is induced by a severe paroxysm of coughing.

The so-called *nocturnal* form of vertigo occurs during the night, when the individual suddenly wakes up, usually after

a very vivid dream, the phenomenon being closely associated with nightmare and usually accompanied by an intensely painful sensation as of falling from a height.

Vertigo is not an unusual symptom in hysteria and in some varieties of anæmia, and it forms part of the picture produced by the pharmacological action of alcohol, when this substance has been swallowed in considerable amount before the usual paralytic phenomena have developed.

When the above etiological factors have been eliminated, it will be found that in the great majority of all cases of vertigo the affection can be traced to some disturbance in the internal ear; hence the symptom-complex known as labyrinthine or aural vertigo (which will be described separately), has the greatest claim to be considered as an independent disease or distinct entity.

The pathogeny of vertigo in general, and of aural vertigo in particular, is a subject of considerable difficulty and uncertainty, since many points are still awaiting elucidation regarding the factors at work in maintaining the body-equilibrium in health.

When the body is in the condition of rest or repose muscular contraction is in complete abeyance, there being no necessity for the operation of any mechanism to control balance or equilibrium. In all other conditions a complicated centric, peripheral, and centripetal mechanism must be called into action in order to secure the maintenance of equilibrium. The location of the centre is more or less a matter of speculation; the information derived from the study of pathological processes within the cranial cavity points to its probable site in the middle lobe of the cerebellum. The function of this centre is to receive centripetal impressions and co-ordinate them so that impulses transferred from it to the cortical and spinal motor centres secure the efficient muscular contraction which maintains the normal body-equilibrium. The various parts of the centripetal mechanism which convey impressions to the cerebellar centre are believed to be: (1) The semicircular canals of the internal ear; (2) the sensory cutaneous nerves; (3) the sensory nerves distributed to muscles; (4) the peripheries of the optic nerve in the retina.

Of these the most important is universally acknowledged to be the semicircular canals. Goltz maintained that they should be regarded as sense-organs, whose function is to ascertain the equilibrium or position of the head in space. According to this authority, 'Every position of the head causes the endolymph to exert the greatest pressure upon a certain part of the canals, and thus excites in a varying degree the nerve-terminations in the ampullæ' (Landois). Injury to the semicircular canals in animals causes disturbance in the body-equilibrium, but does not affect hearing.

Symptoms.—These will be detailed in the following description of auditory vertigo, from which vertigo due to organic brain-disease is only to be differentiated by the presence of the other symptoms characteristic of the special cerebral lesion, as tumour or hæmorrhage, and by the usual absence of marked tinnitus and deafness.

MENIÈRE'S VERTIGO (AURAL or LABYRINTHINE VERTIGO)

This symptom-complex, also known as 'Menière's Disease,' is characterised by the presence of paroxysmal attacks of vertigo or giddiness, associated with more or less persistent deafness and tinnitus.

Etiology and Pathology.—The affection, as implied in its synonyms, is caused by a disturbance of the function of the labyrinth. The nature of the pathological process which affects the terminations of the vestibular branch of the auditory nerve in the semicircular canal is variable. In acute cases the lesion may be a hæmorrhagic one; more frequently it is supposed to be of an irritative nature; whilst many authorities regard it as always the result of a true inflammatory process, due to exposure to cold, syphilis, gout, or rheumatism. Its frequent association with tabes is suggestive of a progressive degenerative change, but it must not be forgotten that many of the eye-paralyses met with in tabes are due to a true neuritis of the ocular nerves, which passes off completely after a brief duration. The symptoms of auditory vertigo may be produced by irritation affecting any portion of the nerve of hearing in its course, either at its origin in the auditory nuclei, in the trunk, or in the peripheral expansions of the vestibular branch in the ampullæ; the very frequent if not constant association of more or less deafness and tinnitus proves that the cochlear branch of the nerve of hearing always also suffers in the irritative or inflammatory lesion. It is a noteworthy fact that the involvement of the nerve in middle-ear disease seldom produces vertigo; but the writer has a case at present under observation in which an extensive hæmorrhage, apparently confined to the middle ear (since bone-conduction was perfect and tinnitus absent) in a diabetic patient, was ushered in with intense vertigo. Not infrequently severe vertigo supervenes upon a thorough syringing-out of the external ear, and this is more likely to occur if the tympanum has been previously perforated. Vertigo is sometimes produced by accumulations of hardened cerumen which press upon the tympanum; foreign bodies in the meatus act in a similar manner. The condition has been found to supervene upon blocking of the Eustachian tube, and it sometimes is the result of spasmodic contraction of the ear-muscles.

When the irritable condition of the peripheral expansions of the vestibular nerve in the ampullæ has become established, it is easily understood how gastric disturbances, ocular troubles, muscular and mental fatigue, severe coughing, vascular changes, and the other etiological factors enumerated upon page 1820, may act as exciting causes and precipitate an attack. It must also be remembered that any blood-condition or angio-neurotic œdema which causes increase in the pressure of the endolymph on the hairs within the ampullæ may induce vertigo; that these causes are, however, seldom operative is rendered likely by the fact that aural vertigo is usually the result of labyrinthine disturbance confined to or preponderating in one ear. The affection is much more common in men than in women, and is comparatively rare before the age of twenty, the majority of the cases occurring after the middle period of life.

Symptoms.—The cardinal features in the symptom-complex of Menière's disease are the paroxysmal nature of the attacks of vertigo and the associated tinnitus and deafness. The attack usually comes on suddenly or abruptly, often with loud noises heard in one ear. These subjective sounds are usually of the nature of an intensification of the continuous or pulsating noises constituting the tinnitus, which is generally more or less persistent between the attacks. Occasionally only are the noises absent at the beginning of the attack; sometimes they are described by the patient as being of the loudness of the sound emitted by a locomotive when getting up steam, or of the roaring character of waves breaking upon a rocky coast. The ear-sound ushering in the attack often follows upon some rapid movement of the body or increase of blood-pressure, as in violent coughing, sneezing, or blowing of the nose; but it may occur during rest, or may awake the patient from a peaceful slumber. Vertigo, if not simultaneously felt, follows within a few moments. Its degree, nature, and duration are most variable; as a rule it might be said to be an intensification or exaggeration of the sensation of giddiness, which in a mild degree is often experienced between the distinct paroxysms. Sometimes the vertigo is so severe as to knock the patient off his feet, as if struck by a powerful blow upon the back of the head. There may be a momentary interference with consciousness, but very seldom is this entirely obliterated; as a rule the mind remains painfully clear and alert all throughout the attack.

More frequently, especially in mild attacks, the vertigo might be described as a sensation of instability, the patient clutching some fixed object whilst feeling as if his body was being rotated, or swayed to either side, or was about to fall in a particular direction. This constitutes the type known as subjective vertigo, and not infrequently the tendency

is to fall or sway towards the side upon which the tinnitus and deafness are most marked, but the exceptions to this are numerous. In objective vertigo there is the sensation as if surrounding objects were in motion. Either type may in rare cases exist alone; in the great majority of instances both subjective and objective vertigo are present at the same time, the patient usually experiencing the sensation that the objects around him are moving in the same direction in which he feels his body to be falling or inclining. Sometimes he experiences a sensation as if the ground was rising up to strike him, and surrounding objects may appear to be rotating like a spinning-top when in motion, so that, in order to prevent himself from falling, he keeps his eyes firmly closed; not infrequently involuntary oscillatory movements may be observed in the eyeballs.

When the paroxysm continues whilst the patient is lying upon his back or after he has fallen to the ground, there is very often the distressing feeling as if his body was sinking through the earth, combined with which is the sensation that the ceiling or walls of the room are in active motion and are about to fall upon him. In this condition he may be compelled to lie flat and motionless for many hours, the slightest elevation of the head or rotation of the body, or even the opening of the eyelids, bringing on a severe exacerbation of the vertigo or precipitating the action of vomiting. The paroxysm may last but a few minutes in mild cases, whilst in severe cases it lasts for hours; and as it passes off vomiting commonly occurs, accompanied by much pallor and sweating. In severe cases the vomiting, like the vertigo, may continue for several hours, the clinical picture closely resembling that observed in a bad attack of sea-sickness, both subjective and objective vertigo alternating with or remaining in association with bilious vomiting and distressing retching. In mild attacks vomiting is very often entirely absent.

The intervals between the attacks vary extremely; paroxysms may occur daily or several times during a single day whilst in other cases the individual attacks may be separated by months or years. It is not a very rare occurrence to witness a typical severe attack of aural vertigo which may never be followed by a recurrence. In the intervals between the attacks more or less tinnitus is constantly experienced (see page 97). This, however, tends in progressive cases to diminish or disappear as the function of the trochlear branch becomes finally obliterated.

Deafness is also nearly always present, but it is not uncommon for this to be so slight as to escape the patient's attention when only one ear is slightly involved. When deafness exists in both ears, almost invariably one organ is more markedly involved than its fellow. There is always

diminished bone-conduction, and this may be the only sign observable upon examination when one ear is but slightly affected. When total deafness supervenes the vertigo disappears, but in some cases the tinnitus may remain.

Diagnosis.—The presence of vertiginous sensations or of actual disturbances of the equilibrium of the body, when associated with deafness in which there is evidence of diminished bone-conduction and when tinnitus also exists, leaves no doubt about the positive diagnosis of Menière's disease. By the presence of these cardinal features the affection is readily differentiated from those cases of ordinary *epilepsy* and *petit mal* in which the attack is ushered in with vertigo, provided the patient is seen in an interval between the attacks. When the patient is for the first time seen during or immediately after an epileptic seizure, there will be the actual evidence or history of complete unconsciousness followed by stupor or sleep, and usually proof of a clonic convulsion is obtainable; vomiting is a rare event in epilepsy, whilst expulsion of the bladder-contents often occurs, and the tongue is frequently bitten.

Prognosis.—The outlook depends upon the nature of the labyrinthine lesion. When this is of a severe acute inflammatory character, according to the authority of Menière, death may result, though few cases are to be found in medical literature where the disease has ended fatally. The physician may confidently assure the patient, who is always seriously alarmed in a first severe attack, that there is practically no danger to life.

The ultimate prognosis is, however, less roseate, some of the subjects of the disease continuing to suffer till total deafness supervenes. This latter is, however, a comparatively rare event, though the loss of hearing may become complete in one ear. Under careful treatment, few chronic maladies respond more satisfactorily to medication.

Treatment.—The indication is to reduce the hyperexcitability of the peripheral terminations of the auditory nerve in the semicircular canals and at the same time to render more stable the equilibrium of the co-ordinating centre in the cerebellum. Both indications are met by bromides in full doses, the treatment being practically identical with that of epilepsy, but much more satisfactory results are obtainable than are to be expected in the latter disease. Quinine, salicylates, digitalis, and, more recently, pilocarpine have been extolled, but 1 dr. to 1½ dr. of sodium bromide given daily in divided doses meets all requirements. Though gastric, cardiac, and other so-called varieties of vertigo are probably primarily due to disturbance in the labyrinth (since dyspeptic and valvular affections may act as exciting causes), treatment is also clearly indicated upon

the lines recognised as suitable to these affections. In an empiric manner counter-irritation behind the ears may be employed, and is often most beneficial. The tinnitus often completely disappears under the bromide-treatment.

VITILIGOIDEA, XANTHELASMA, or XANTHOMA

These are the titles given to the condition in which yellow growths, in the form of plaques or nodes, are found in the skin covering different parts of the body.

The affection is not uncommon when it affects the eyelids alone, in which case the lesion is of the flat form, consisting of a thin plate of new-growth (*X. planum*), which first shows itself usually in the upper lid of each eye near the inner canthus, and gradually extends outwards till sometimes the skin of the lid is invaded for a half or more of its extent. The plate is embedded in the corium; the skin is but slightly elevated and appears to be quite normal on the surface to the touch and sight. Morris likens the appearance of *X. palpebrarum* to a piece of chamois leather let into the skin. Sometimes a similar plate appears in the lower lid, so that the entire margin of the orbit is involved in one continuous patch, but more frequently by a series of smaller plaques or plates which lie alongside each other. The disease is always bilateral and usually appears after forty, being met with more frequently in women. In many instances an hereditary history is obtainable, and often a history of migraine or jaundice is found. After extending for a time, the growth of the plate stops and usually remains unaltered and permanent.

In rare cases numerous plates are found embedded in the corium in other regions, constituting the variety of xanthoma known as *X. multiplex*. These are always symmetrical, and may be found upon the palmar aspect of the hands and in the soles of the feet and in the skin over joints and over the buttocks; the mucous membrane covering the different parts of the mouth, gullet, respiratory tract, and bile-passages may be involved. In this multiplex variety the growths are found to be both of the flat or plate type and of the nodular variety (*X. tuberosum*). The latter type of growth is seen chiefly in the skin over the knuckles, knees, and elbows. In the rare examples of xanthoma multiplex occurring in children the eyelids are stated by Morris to be always free from the growths, and, like the palpebral variety, an hereditary history is often obtainable in both children and adults; in the latter the condition is very frequently, but not invariably, associated with chronic jaundice.

Pathology.—The pathology of the affection is doubtful; some authorities regard the palpebral variety as of the nature of a degenerative lesion. The usually accepted view of the nodular variety is that the lesion is of the nature of a chronic inflammation, as first held by Chambard. The xanthoma-cells constituting the growth are supposed to be derived from the connective tissue; they contain droplets of fat and closely resemble those found in atheroma.

Morris described a rare form of xanthoma which he found in patients suffering from glycosuria. This *X. diabeticorum* is differentiated from the multiplex variety by the conical shape of the yellow nodes, which are surrounded by a raised red area which gives the affection a close resemblance to acne; they come out in crops and disappear in a few weeks. Any part of the cutaneous covering may be involved, but usually they occupy the extensor aspects of the limbs, the region of the loins and lower part of the abdomen and buttocks, and occasionally the mucous membrane of the mouth; the eyelids usually escape, and sometimes considerable itching may be associated with the affection.

A very rare form of xanthoma associated with the name of Balzer is recognised, in which the yellow raised patches resemble the ordinary type in appearance, but its histological peculiarity lies in obvious changes observable in the yellow elastic fibres.

Xanthoma, except the diabetic variety, which yields to dietetic treatment, is uninfluenced by medication; when the nodes or plates cause deformity or unsightliness excision may be recommended, but this is very seldom necessary.

WHOOPING-COUGH

This, known also as Hooping-Cough, Chin-Cough, and Pertussis, is an acute infectious disease characterised by the presence of more or less bronchial catarrh, associated with severe paroxysmal attacks of convulsive coughing, each of which is followed by a remarkable crowing inspiration pathognomonic of the affection.

Etiology.—The disease commonly occurs in epidemic form, though sporadic cases are continually being met with. Infants at the breast are often affected, but the majority of the cases occur between the first and second dentitions. Though essentially a disease of childhood, the affection is occasionally to be met with in the middle or advanced period of life. One attack almost invariably confers a lifelong immunity, and in the rare cases occurring late in life a history of an attack in childhood is practically never met with. For many months, or sometimes for a year, after the attack in the

child has passed away a subsequent attack of bronchial catarrh may have associated with it a cough in which the shrill whooping inspiration is occasionally heard. This has led to the erroneous belief that second attacks are not very uncommon before the end of the seventh or tenth year. A somewhat similar phenomenon is occasionally witnessed in malarial subjects who, under trivial urethral or other peripheral irritations, may exhibit the characteristic rigors, and hot and sweating stages of the original disease, which has left some very marked condition of susceptibility of the nerve-centres long after the disappearance of the pathogenic organisms and of their toxins.

Epidemics of whooping-cough occur much more frequently in the late autumn or early spring months, and they often follow or accompany epidemics of measles, and sometimes are observed also to be in a similar manner associated, though less frequently, with epidemics of scarlet fever. During an outbreak it is frequently noticed that children suffering from epidemic or other form of bronchial catarrh are more liable to catch the infection.

There cannot be a doubt that the disease is highly infectious, being in the great majority of cases transmitted by personal contact; but it is equally certain that the specific germs may be transmitted by a third person and by means of fomites attached to clothing, though these latter methods of conveyance are so seldom witnessed that their potentiality has been doubted by some competent authorities.

The sputum is believed to contain the virus, and the explosive nature of the coughing paroxysms affords an obvious explanation of the highly infectious nature of the disease, since the mucous secretion from the bronchial and nasal tract is ejected for a very considerable distance from the patient's body in the form of a fine spray containing droplets of mucus. Though many observers have described various micro-organisms discovered in the sputum, it cannot be accepted that any of these should be regarded as the specific microbe of the disease, even when convulsive coughing has followed their introduction into the blood or bronchial tract of animals, since it is known that many different streptococci and diplococci may produce this effect. The infectious nature of the bronchial secretion has been proved to be present at a very early stage of the disease even before the development of the characteristic whooping inspiration, and the danger of infection remains, though it is believed in a diminished degree, till after the convalescent stage of the disease has been entered upon. An influenza-like bacillus has been described by Spengler, Davis, Jochmann, and others, which has been found by Wollstein to answer the usual agglutination-test.

Morbid Anatomy and Pathology.—The changes observed after death from whooping-cough cannot be regarded as in any sense characteristic. They are such as are produced by the various complications so often witnessed during the progress of the malady, as collapse of the pulmonary lobules often associated with pneumonia of the bronchopneumonic type, and not rarely with a considerable degree of compensatory emphysema. The usual changes characteristic of bronchial inflammation are invariably present, the lining membrane of the tubes being deeply injected, swollen, and infiltrated with leucocytes, and more or less enlargement of the tracheal and bronchial lymphatic glands is almost always present. The tubes contain a considerable quantity of sputum similar to that expectorated during life.

Considerable attention has been directed to the frequency with which ulceration of the mucous membrane in the region of the frænum linguæ has been observed, and although this may be regarded as a valuable diagnostic sign during life, it cannot be accepted as of any pathological significance, being probably the result of mechanical injury to the part caused by the frænum being propelled against the sharp edges of the lower incisor teeth in the violent explosive coughing-fits which cause the tongue to be projected from the floor of the mouth. The blood-condition will be referred to later on.

The pathogeny of the affection is probably explicable upon the hypothesis that the toxins of the specific microbe produce an irritable condition of the bronchial surface, with which is associated a similar state of the pneumogastric peripheries, which latter explains the element of laryngeal spasm always present. Eustace Smith draws attention to the general condition of nervous unrest and excitability, and rightly regards this, and also the vomiting, as probably the result of centric influences more than of simple reflex phenomena. The theory that the paroxysmal cough is due to the pressure of enlarged bronchial glands upon the laryngeal nerves is no longer accepted as an explanation of the phenomena of pertussis.

Symptoms.—The incubation-period of whooping-cough is regarded as varying from about five to fifteen days. The progress of the disease is usually easily divided into two well-marked periods—viz. the catarrhal and the paroxysmal or spasmodic stages, the latter imperceptibly passing into a third stage, which has been described as the period or stage of decline.

The Catarrhal Stage.—The first symptoms of the affection show themselves in a simple bronchial catarrh associated usually with some implication of the upper respiratory tract. There is slight pyrexia, mucous discharge from the nose, with sneezing, some suffusion of the conjunctivæ, and a dry and

often painful, somewhat spasmodic cough, especially evident at night. As in an ordinary feverish cold, there is usually some malaise, loss of appetite, and listlessness or languor observable in young children; older patients may complain of headache. This condition may last for one or two weeks, during which period there is usually no sign or symptom to arouse suspicion, save the increasing tendency of the cough to become incessant as evening or night approaches; though expectoration may be free, it does not afford the relief which is usually apparent in cases of ordinary bronchial catarrh.

The Paroxysmal or Spasmodic Stage.—This may be abruptly ushered in by the characteristic crowing inspiration, which when once heard settles the diagnosis conclusively. More frequently some of the paroxysmal coughing-fits during the later stages of the catarrhal period are followed by a noisy inspiration in which the experienced ear is usually able to detect more or less of the elements of the whooping sound. The full development of the spasmodic stage is characterised by a series of short, quickly recurring, hacking coughs, which expel all the air out of the chest and cause more or less transient cyanosis. The inspiratory muscles are next brought suddenly into violent action, drawing the air into the lungs through the partially closed laryngeal orifice with a characteristic loud crowing sound or whoop. The preliminary series of expirations constituting the paroxysm of coughing are not broken or interrupted by any attempt at inspiration till the advent of the whoop. During the coughing-fit much clear, tenacious, mucoid sputum may be brought up, and the phenomenon may be completed by the ejection of the stomach-contents, which always show a very considerable quantity of mucus, the source of which is mainly swallowed sputum, though there is obviously increased gastric secretion also mixed with the food. The bladder and bowel contents are not infrequently expelled at the same time.

After a few such typical paroxysms the child learns to recognise the advent of succeeding attacks; his features are observed to become set in his attempt to restrain the convulsive expirations, and he soon finds that by seizing upon some firm object with both hands he can assist the expiratory efforts. The clinical picture thus produced is unmistakable, and no difficulty in diagnosis can occur when the physician is present to witness a seizure. Often an examination of the throat brings on a severe paroxysm, especially if the patient resents the interference, and the firm depression of the tongue by a spoon or depressor is certain to cause a series of kinks, followed by the crowing inspiration; mental and emotional excitement of any kind usually brings on an attack when the disease has become established.

The pulse during the paroxysm is small and exceedingly rapid ; the congestion of the face and head may pass into that of deep cyanosis suggestive of complete asphyxia in severe cases, and if the chest be exposed at the time signs of cardiac engorgement may be obvious. The lower part of the thorax will be found retracted during the inspiratory act owing to the obstruction to the access of air caused by the laryngeal spasm. The intensity of the congestion of the vessels may leave petechiæ or hæmorrhagic extravasations visible under the conjunctival membrane or in the swollen skin of the eyelids and forehead ; and not infrequently blood may pour out from the nasal orifices or even from the eyes, ears, and mouth.

The violence of the expiratory efforts may be manifested by the production of hernia and prolapsus ani, and sometimes the paroxysm may eventuate in an attack of general convulsions.

Physical examination of the chest in the intervals between the attacks may reveal nothing abnormal ; usually, however, the signs of a catarrhal condition of the larger and medium-sized bronchial tubes will be present.

There are sibilant and sonorous rhonchi with bubbling sounds usually heard about the bases of the lungs behind. Occasionally some evidence of emphysema may be detected, and as a rule, in the absence of pulmonary complications, a hyper-resonant note may be elicited by percussion.

The attack leaves the child very much exhausted, and often more or less drowsiness or deep sleep may follow. In mild cases, in the intervals between the paroxysms the patient may regain the normal condition and be able to run about and play as usual ; the degree of improvement will obviously depend upon the frequency of the paroxysms. The number of the attacks varies very greatly—from half a dozen or less to fifty or more during the twenty-four hours may be observed ; and when vomiting is a marked feature the exhaustion resulting from the ejection of food will seriously threaten life.

The duration of the disease varies almost as widely as do the number and degree of severity of the paroxysmal seizures, even when no complications are present.

The Stage of Decline.—This sets in usually during the fourth week from the commencement of the catarrhal stage, when the paroxysms show signs of less severity and the intervals between them lengthen, some of the kinks of coughing not being accompanied by the whooping sound. In this stage the bronchial secretion is free and easily brought up by coughing, but, like the fits of whooping, it, too, gradually fades away almost imperceptibly from day to day along with the cough.

A very mild attack of the disease may safely pass through its different stages and end in convalescence by the end of the third week ; but much more frequently the duration of the affection exceeds twice this limit, and cases are by no means uncommon where the duration may be protracted beyond the eighth or tenth week.

Complications.—The most common and important of these are the pulmonary lesions, especially bronchopneumonia or capillary bronchitis, which is so liable to prove fatal and which may supervene at any stage of the disease. The whoop usually entirely disappears as soon as the fine tubes and lobular tissue become involved ; the symptoms deepen suddenly, dyspnœa and cyanosis continuing all through the intervals between the paroxysms of coughing. The cough tends to become almost continuous and the usual signs and symptoms of suffocative catarrh develop, and, according to Eustace Smith, there may be dangerous laryngeal spasm, even when no whooping sound is audible. It must be remembered that very young infants may never whoop throughout the attack.

True croupous or lobar pneumonia is a much rarer complication than the lobular variety. When it occurs it may run its short course in the ordinary manner, being apparently little influenced by the pertussis, and only accentuating the distress of the breathing to a moderate degree, whilst the lobular pneumonia often changes the entire clinical picture owing to the amount of asphyxia induced by the blocking of the fine tubes and air-cells scattered throughout all the lobes of both lungs.

Collapse of the lung is the formidable complication to be dreaded in infants and very young children. It may occur after any unusually severe paroxysm of coughing which empties the lung of air when the inspiratory power of the infant is reduced by the exhaustion following frequent vomiting. The advent of this complication is heralded often by a convulsion, especially in rachitic children, and it may be followed swiftly by death, even before cyanosis and symptoms of asphyxia have time to develop, the main symptoms being those of extreme general collapse, with pallor of the face and coldness of the extremities. Collapse of the lung is also not an infrequent accompaniment of the bronchopneumonia of older infants and children. In rachitic subjects sudden death may result from spasm of the glottis.

Pulmonary emphysema rarely gives rise to any obvious symptoms, but occasionally in a severe paroxysm of coughing rupture of a cluster of distended air-vesicles may occur, the extravasated air finding its way into the mediastinum and in rare instances causing general subcutaneous emphysema, which may travel over a considerable area of the body.

Pneumothorax may sometimes be produced in a similar manner.

Acute pleurisy is a rare complication which may end in empyema.

The enlargement of the bronchial lymphatic glands is probably always present and cannot therefore be strictly regarded as a complication, but occasionally it may be so extensive as to produce pressure-symptoms which greatly embarrass the respiration, and at a later stage cascation and suppuration may follow.

Complications involving the nervous system have been incidentally referred to; the most formidable of these are convulsions, which, though not necessarily fatal, should always be regarded as of serious omen, since they may be due in some cases to serious organic changes in the brain or cranial cavity. Ordinarily the convulsion is, like that of the common infantile type, due to the increased excitability of the motor centres, but occasionally it may be caused by engorgement or extravasation of blood in the cortex of the brain, or may be the result of subdural hæmorrhage, sinus thrombosis, or embolism. In these last-mentioned conditions aphasic, hemiplegic, or paralytic symptoms may follow, and in some cases the latter symptoms may permanently remain. It must also be remembered that the convulsive seizure may be a rachitic manifestation or the result of an attack of pneumonia or of collapse of the lung. Meningitis is a rare complication, and when it occurs it is likely to be of tubercular origin. Various forms of paralysis—centric, bulbar, or peripheral—may occasionally follow, even in cases where no convulsions occur.

The vomiting frequently present in the paroxysmal seizures may be so severe and continuous as to rank as a serious complication of the disease, and this is especially likely to be dangerous when diarrhœa is present. In this latter case the little food which the stomach retains may be hurried through the alimentary canal before absorption has time to occur, and the patient will probably soon succumb to marasmus, the result of acute starvation.

Fever, when present, is to be regarded as the result of the pulmonary complications, since after the early part of the catarrhal stage the temperature-chart generally exhibits an apyrexial type of disease.

As pertussis so frequently is associated with measles and, to a less extent, with scarlatina, the progress of the disease is very generally associated with the complications peculiar to these affections, as middle-ear disease, albuminuria, &c.

Sequelæ are not uncommon, and the most important of these is pulmonary phthisis, which in a variable proportion of cases originates in the damage to the pulmonary substance caused by

the bronchial pneumonia, the interstitial changes preparing a nidus for the development of the tubercle bacillus. In some cases acute general tuberculosis has been seen to follow in the convalescent stage. Asthma and pulmonary emphysema, with more or less deformity of the thorax, are more common sequelæ. There is no proof that permanent valvular disease has its origin in pertussis, though many experienced observers believe that the strain upon the valves during the violent expiratory efforts sometimes leads to lasting injury to the valve-segments, which may show itself at a late stage.

Diagnosis.—In the early stage this is usually impossible, but a valuable aid to diagnosis at this period is found, according to Churchill, in a careful blood-examination. Leucocytosis is nearly always to be found at all stages of the disease, and in a large proportion of cases (85 per cent.) during the catarrhal period, before the advent of the whoop, a well-marked lymphocytosis can be detected. Many observers lay stress upon the remarkable light colour of the urine, when considered with its constantly high specific gravity, and others attach importance to the frequent presence of a heavy white precipitate of uric acid. Perhaps the most suspicious sign or symptom at this early stage is the very marked tendency of the cough to become more aggressive and severe as night approaches, and its *explosive suddenness* may be a marked characteristic before the whoop develops. At a later stage the presence of the whoop is distinctive, and the physician may elicit this always by thoroughly depressing the tongue; indeed, in the majority of children it will be found usually sufficient to threaten an examination of the throat, as the spasmodic seizure is easily induced by the dread which the sight of the tongue-depressor or laryngeal mirror excites. The presence of Riga's sublingual ulcer involving the frænum is a valuable diagnostic point, but it is never found in the catarrhal stage, since it is produced only after the violent convulsive cough causes the under-surface of the tongue to be projected above the sharp edge of the incisor teeth; consequently it is not observed in infants at any stage of the disease prior to the eruption of the central incisors. Though the whoop may practically be regarded as pathognomonic of the disease, an identical sound has been reported by Parkinson to occur in fibrosis of the lung, accompanied by the presence of enlarged bronchial glands lying in the space close to the bifurcation of the trachea, causing pressure upon the left vagus nerve.

Laryngismus stridulus may be mistaken for the whoop of pertussis, but its intimate association with rachitic signs and the absence of the series of short, violent, explosive coughs which always immediately precede the whoop of pertussis are characteristic; moreover, vomiting, bronchial catarrh,

and the expulsion of bladder and bowel contents do not accompany the phenomenon. The same remarks apply to the cough caused by *adenoid* growths and *enlarged tonsils*. McKisack states that 'a night cough, paroxysmal in character, and resembling whooping-cough, but not occurring as a rule during the day, and usually observed in children, is due to enlarged tonsils, adenoids, or other irritation in the nose and naso-pharynx.'

Enlarged mediastinal lymphatic glands pressing upon the nerves supplying the laryngeal muscles, as already mentioned, may cause a cough practically identical with that of pertussis, but the history of the case and its duration will usually settle the diagnosis. There will be absence of the almost characteristic general nervous hyper-excitability, vomiting, relaxation of the sphincters, constant oedema of the eyelids, bronchial catarrh, &c., peculiar to pertussis; moreover, such marked enlargement of the bronchial lymphatic glands as is necessary to simulate pertussis must be of tubercular origin, and other evidence of scrofula or tubercular adenitis is sure to be found elsewhere, if carefully sought for.

At a later stage the bronchial and pneumonic signs, when persistent, are certain to arouse the suspicion of *phthisis*; and it must be remembered (if the child is first seen by the physician when this complication is present), the characteristic whoop may be entirely absent. In cases at this stage, if no history is obtainable, mistakes are liable to be made; and this is especially true when pertussis has followed measles, as the bronchopneumonia is then very liable to run a slow course, and to exhibit its lingering presence long after the other symptoms have cleared off. It is in such a combination of events that Calmette's tuberculin instilled into the conjunctival sac will often afford conclusive evidence of the existence of latent tubercular disease.

The physician is liable to be taken unawares in the diagnosis of whooping-cough occurring later in life owing to the extreme rarity of the affection after the period of childhood; the only difficulty in the diagnosis of such cases exists in the chance of the patient never having a paroxysm of coughing in the physician's presence. The history or description of a cough possessing sudden explosive features, especially when chest-signs are vague or absent, should always arouse suspicion of pertussis.

Foreign bodies in the trachea or larynx may cause explosive cough accompanied by laryngeal spasm simulating pertussis, and when a previous bronchial catarrh has existed the condition may for a short time be mistaken for pertussis.

An interesting phenomenon has been so often observed in the progress of the disease that there cannot be a doubt about its acceptance, and it should be remembered in dealing

with the affection in infants—*i.e.* the disease is sometimes completely arrested during the evolution of the vesicle after the operation of vaccination.

Prognosis.—Pertussis is a much more serious and fatal affection than is usually realised by the parents of poor and ill-fed children. The gravity of the prognosis depends largely upon the age of the child. Voelcker's recent statistics show a mortality of about 28 per cent. ; 75 per cent. of the fatal cases were in infants under two years of age. Larger statistics prove that over 95 per cent. of the fatal cases occur in children under five years of age. These figures show the vital importance of preventive measures, since every year during which the child can be shielded from the danger of infection will afford an increasing degree of safety when the disease does appear. In this respect the prophylaxis of pertussis is upon a par with that of scarlatina. Collapse of the lung in young infants, and bronchopneumonia in older children, are the main causes of death. In Voelcker's cases 50 per cent. of the patients, in whom bronchopneumonia supervened, died, and 10 per cent. of the total mortality was due to pulmonary tuberculosis.

Treatment.—In the author's 'Dictionary of Treatment' (4th ed.), pp. 700–710, the details of the therapeutics of pertussis are fully discussed, and only the principles which should guide the treatment need be here mentioned.

Owing to the enormous importance of preventing the spread of the disease to young infants and children under five years of age, rigid prophylactic measures should be resorted to.

Isolation is the first question to be settled. This is to be decided by the peculiar nature of the patient's surroundings, and, as it generally will be required for eight or ten weeks under ordinary circumstances, there is no use in beginning a system of isolation in a half-hearted way, nor is there any use in insisting upon it where it is almost certain to break down in a few weeks. Moreover, no plan of isolation should be accepted which will confine the patient to one room during the entire illness. As a rule, in this country the patient's parents, who think that they know a good deal about such a common disease, refuse to carry out a system of rigid isolation. The writer's practice is to warn the patient's relatives of the danger which might arise if some weakly child or infant of the family were to catch the contagion, and to urge upon them the advisability of placing two rooms in the house at the disposal of the invalid, and advise that the others should be sent away.

The child should have one large well-ventilated sleeping and one play room, especially in the winter time, and at all times when possible. This is a matter of more importance than nurses and parents can be made to see. The air of the

room can be medicated by the methods to be mentioned later on. The temperature of the chamber is an important matter ; in severe cases requiring to be kept indoors, the temperature of the room should be *kept* at 65° F., but it is a serious mistake to do this and permit the child to go out into the cold air. The second room in these cases can be used as an 'intermediate.'

The next point to be settled is the one of permitting the child to go outdoors. Moderately strong children, even in smart attacks, not only are safe, but are much better for being out all through the attack. *Plenty of fresh air* is really of more importance than medicine in a long illness like whooping-cough. Of course, in severe weather, with rain and cold, or during the prevalence of the spring east winds, or in the presence of any bronchial complications, outdoor exercise must be forbidden.

Diet is of great importance, and, in the management of weakly young children, will turn the scale for or against recovery. The usual diet which experience has proved to agree best with the child is to be continued, provided it be wholesome and highly nutritious. A mixed and varied diet does best with grown-up children. After the paroxysms have become thoroughly established and the appetite begins to fail, the patient must be coaxed to take milk or other nutritious fluid nourishment between meal-times.

After a time, when the paroxysms are attended by vomiting, the critical period in the dieting arrives. The nurse should be directed to withhold food for a short time in the presence of a threatening or expected attack of coughing, and to aim at having the child fed as soon as possible after an attack, so as to permit the food to be as long as possible in the stomach before the next turn of vomiting. By carefully attending to this hint lives may be saved.

Hard indigestible food in the stomach, as nuts, green fruits, unripe apples, &c., will increase the laryngeal spasm, and portions of unchewed beef and potatoes may give serious trouble during the act of vomiting. Hence, upon the whole, a liquid diet is to be preferred for small children, or the ingredients should be carefully comminuted.

Clothing should be warm, and so arranged as to prevent overheating at one time and chills at another, an even temperature and the avoidance of draughts being desirable. Light flannel underclothing is essential, except in very warm summer weather.

During the paroxysms young children should be lifted on to the nurse's knee or lap, and every constriction about the neck or chest should be removed. Naegeli states that the paroxysms may be arrested by simply pulling the lower

jaw downwards and forwards, and this is effective also during sleep.

As regards drugs, the list is a long and tedious one to discuss. Most physicians find themselves after a time settling down to the routine employment of one or two simple expectorant agents. Active or heroic medication is to be condemned. Since the improvements in our knowledge of bacilli and the part played by them in the various infectious diseases, many germicides have been tried in the treatment of whooping-cough, but to none of these can a specific action be fairly attributed. The plan of treating the disease by inhalations can hardly be said to have proved a success, though antiseptics of a volatile and unirritating nature certainly appear to modify and sometimes to shorten the duration of the disease.

Carbolic acid in the form of a spray (1 in 100) may be inhaled by the patient three or four times a day, or a stronger spray may be diffused frequently through the air of the room. The plan of forcing young children to submit to such solutions being sprayed *directly* into the mouth is not to be recommended. They often increase the frequency and severity of the paroxysms. Yeo insists upon the great value of carbolic-acid *inhalations*. In the sick-room he keeps by the fire a large iron dripping-spoon for vaporising carbolic acid, which should be used till the air of the room becomes unpleasantly strong with the fumes. At the same time he uses a spray of 1 dr. glycerin of carbolic acid, 10 grs. bicarbonate of soda in 1 oz. hot water, to be sprayed in front of the patient's mouth and nose. In addition he causes a warm 5 per cent. spray of carbolic acid to be diffused through the air, and sometimes in young infants brushes the pharynx and larynx with glycerin of carbolic acid.

No objection can be made to the air of the sick-room being impregnated with a volatile antiseptic like turpentine poured over the surface of hot water. Terebene, eucalyptus oil, peroxide of hydrogen, &c., may be similarly used, and they are of the greatest benefit where several children are confined in one room in severe weather.

Creosote is praised by Godson, who gives it by the method of continuous inhalation, the drug being sprinkled upon cloths hung up in the sick-room; and he states that it produces an immediate effect upon the paroxysms, which are rendered less frequent.

Little can be said for the methods suggested for cutting short the disease by the application of various solutions to the fauces or larynx, and the strong solutions of cocaine recommended are dangerous, as well as useless in most cases.

Resorcin spray, 2 per cent., is safer, and very glowing reports of its use are published. Dawson Williams states that

from the local application of a 2 per cent. resorcin-solution to the throat he has seen more striking results than from any other method of treatment. Richardson extolled peroxide of hydrogen internally and as an inhalation. Strong solutions of caustic or nitrate of silver are still painted over the fauces by some.

As regards drugs which are supposed to control the severity and diminish the frequency of the paroxysms, almost every known antispasmodic agent has been recommended. In the early catarrhal stage expectorants are clearly indicated; the remedies which act upon the bronchial membrane and diminish the adhesiveness or viscosity of the sputum undoubtedly lessen the risks of collapse of the air-cells and minimise the dangers of bronchopneumonic complications at a later stage. The best routine at this period is the administration of a simple expectorant of the ipecacuanha class combined with aromatic spirit of ammonia and small doses of bromides. As soon as the characteristic whoop has developed, bromides may be safely given in full doses, and continued all through the paroxysmal period. The addition of antipyrin to the bromide mixture is usually all that is required in the great majority of all mild cases of the disease. When the convulsive coughing-seizures are severe and frequent there is no drug so universally applicable as chloral hydrate. It may be given every two hours in doses of half a grain for every year of the child's age, and rarely is a larger dose indicated for continuous administration. When convulsions supervene, the combination of chloral and bromides may be safely pushed. Morphia and belladonna or atropine are extolled by many physicians, but the danger of the first-mentioned drug, when pulmonary collapse and bronchopneumonia have supervened, is a real one, and in the writer's opinion the mortality rate is seriously increased by its routine administration.

In very severe paroxysms chloroform-inhalations may be resorted to, but obviously this method of relief cannot be employed continuously. Berguete discards all antispasmodic drugs, relying entirely upon warm or hot baths (102-107° F.) every six, eight, or twelve hours, and claims for this hydropathic method that it sometimes causes the disease to abort when applied in the catarrhal stage, and that in the paroxysmal stage it modifies the severity and duration and frequency of the attacks more effectually than any other procedure. Similar claims have been advanced for quinine in large doses, and also for euquinine. Steckel recommends the administration of the latter drug in suppository form, in 3-gr. doses to infants. In the experience of the writer quinine in large doses is not free from danger, as it seems often to dry up the bronchial secretion, rendering this more viscid and

adhesive, consequently increasing the danger of pulmonary collapse.

In the stage of decline the question of change of air should be considered, and the old plan which was so frequently resorted to of bringing the patients to a coal-gas manufactory in order that they might inhale the fumes of the gas-lime was often followed by marked benefit. Mohn carries out this principle at home by removing the patient from his sleeping-room in the morning in order that the apartment may be thoroughly fumigated and disinfected by the fumes of burning sulphur, with the doors and windows closed for at least five hours. After free ventilation the patient is permitted to return to the room in the evening. Marked benefit and relief to all the symptoms have been frequently observed to follow this method, and often it seems to suddenly cut short the duration of the attack.

The various complications as they arise are to be treated upon recognised therapeutic principles, and every precaution should be taken to minimise the danger of the supervention of pulmonary phthisis in weakly children by open-air living, over-feeding, cod-liver oil, and the agents found valuable in the sanatorium treatment of pulmonary tuberculosis.

Dr. J. McCaw maintains that the period of quarantine should extend from six weeks to two months from the onset of whooping before the child is permitted to return to school.

WRITER'S CRAMP. (See under CRAMP.)

YAWS

This is a specific tropical disease characterised by the presence of a primary sore arising, after a variable period of incubation, at the site of inoculation, succeeded by a secondary stage marked by constitutional disturbances and skin-manifestations, and sometimes followed by tertiary lesions or sequelæ.

The disease is known by a variety of names, some of which are local, as Frambœsia, Paranghi, Pian, Coco, Amboyna Button, &c. It derives its chief interest from the controversy which is still vigorously maintained regarding its relationship to syphilis.

Etiology.—Yaws, though a tropical affection, has some marked peculiarities of distribution which are suggestive in connection with the problem of its supposed syphilitic nature. Thus it is very rare in India, where syphilis flourishes, and is becoming rare in British Guiana as syphilis has become more

common there. It is very prevalent in Ceylon, Java, Madagascar, the West Indies, and the western coast of Africa.

Though no race is exempt, the disease is most frequent amongst the Africans, and in mixed populations only those uncleanly Europeans as a rule who mix with the natives suffer from yaws. The disease is rare after thirty-five, the majority of cases occurring in young children and very young adults. It is never congenital or hereditary.

The specific lesions have been investigated by Castellani, and his report in the January number of the 'Journal of Tropical Medicine' for 1906 contains a description of the different forms of spirochætae found in the scrapings from the cutaneous ulcerations. No less than four apparently separate varieties or species of these protozoan organisms have been found in the disease. One of them has only been detected in the lesions which were not ulcerated, and this, called *spirochæta pertenuis*, is claimed to be identical with the specific microbe of syphilis—*spirochæta pallida*, or *treponema pallidum*, as it is also designated. If the identity of these can be demonstrated, the question must be regarded as settled that yaws is some modified form of syphilis.

In the meantime the following apparently radical differences are accepted as present, which go to prove that the affections are distinct entities. Thus the virus of yaws can be transmitted from one ape to another by inoculation; but syphilitic apes remain susceptible to yaws, and apes inoculated with yaws can be rendered syphilitic by the inoculation of true syphilitic virus. In the same way syphilitic patients have been known to contract yaws, and *vice versa*. The virus of yaws is auto-inoculable, unlike that of syphilis; the disease is never transmitted by heredity as in syphilis; the skin-lesions show no polymorphism; in the secondary and tertiary lesions the characteristic syphilitic changes are not found in the coats of the blood-vessels; there are no evidences of visceral mischief; and Hutchinson's teeth and keratitis are not found in yaws. A further vital difference is observable in the nature of the secondary eruption in the two affections, which will be referred to later on.

Symptoms.—The disease is transmitted by contact of the mucous membranes, as in sexual intercourse and kissing, and by contact of an abraded skin-surface with the virus; though it is held that a breach of surface is not absolutely necessary for the transmission of the disease. The primary sore appears as a raised red papule or fungoid excrescence after an incubation-period of from one to less than three months, the site being usually extra-genital—viz. on the lips, tongue, perinæum, groins, or other region. The flat papular excrescence is capped within about a fortnight by a dry scab, underneath which a small ulcer with raised edges and fungoid

granulations is found to exist. No induration is observed to surround it, the margins rising abruptly from the healthy skin.

The *secondary* period follows the first appearance of the primary sore in about four or five weeks, and is ushered in by pyrexia and other constitutional disturbances at a variable interval, after which the so-called secondary eruption appears. This consists of a series of small fungating papules, which are distributed over a considerable portion of the surface of the body. These pass through the same stages of scabbing and ulceration as the primary sore, and eventuate in the characteristic raspberry-like granulations which have given the name of Frambœsia to the disease. A few of these granulating papules may be seen about the inside of the margin of the nose, and even in the mouth and around the anus ; but there are no early throat-ulcerations or mucous patches on the tonsils, as observed in syphilis. Marshall thinks that the late pharyngeal ulceration met with in yaws may be due to superadded syphilis.

As regards the *nature* of the eruption, two very important facts must be remembered : not only is it monomorphous, as already mentioned, and hence unlike the syphilitic rash, but it is non-symmetrical and possesses the same features as the primary sore and passes through the same phases, the fungoid 'mother yaw,' as the natives call the primary sore, being reproduced in each flattened raspberry-cluster of granulations seen in the secondary eruption. This is obviously a very different condition of affairs from what is seen in typical syphilis, in which the physical characters of the eruption differ most materially from those of the primary sore. Iritis and alopecia and mucous patches on the tongue and anal region do not occur. The raspberry-papules are always accompanied by considerable itching, and their appearance is preceded by numerous white furfuraceous itching patches on the trunk and limbs. The papules in a few weeks tend to lose their raspberry-red hue and become bleached in appearance. In children (who constitute the majority of patients) they last from three to six months, whilst in adults their average duration is about twice as long ; the healing of the ulcerated surface is afterwards marked by a white scar. Fatal anæmia, septicæmia, and even pyæmia may follow when the ulcerations are deep and extensive. The sequelæ correspond within certain limits to the tertiary manifestations seen in syphilis, and consist of deep ulcerations, which may extend down to the periosteum of the bones and may be accompanied by nodular infiltrations of the subcutaneous tissue. Occasionally nodal swellings are observed upon the long bones as in syphilis. There is often extensive destruction caused by deep ulceration of the pharynx and palate, and

sometimes considerable permanent thickening of the bones and joint-changes are observable. One attack of the disease usually confers immunity, but second attacks have been met with occasionally.

Diagnosis.—The only disease likely to be confounded with yaws is syphilis, and the differential features have been already emphasised. Some examples of the affection may be confounded with leprosy, but the absence of the specific bacillus of the latter disease will clear up the diagnosis.

Prognosis.—Death only occurs in the very severe and rare types of the disease where the ulcerations are deep and extensive; and often in these cases there remains the doubt of a previous or superadded syphilitic infection.

Treatment.—Yaws yields to mercurial and iodine treatment in the same manner as a mild case of syphilis does; but the virtue of mercurials is not demonstrated in the early stage of the disease, and Morris states that they always aggravate the symptoms when given at this period. *Large* doses of iodides combined with small doses of mercury afford the best results in all cases. The ulcerations caused by the breaking down of the papules require the most stringent cleanliness and antiseptic treatment, the best local applications being iodoform or weak perchloride lotions.

YELLOW FEVER

This is an acute specific disease confined to warm climates, transmitted through the bite of a mosquito, and characterised by a brief continuous fever, jaundice, black vomit, albuminuria, and other serious hæmorrhagic features.

Etiology.—The disease is never absent from its endemic focus, which includes the islands comprising the West Indies, the seaports along the shore of Mexico in the Mexican Gulf, Brazil, and Western Africa along the coast of Senegambia. From these regions it periodically extends in epidemic form southwards among the Spanish-American ports to Monte Video and Buenos Ayres, and northwards amongst the ports on the Atlantic coast of the United States to Philadelphia, and sometimes to Boston and New York; and westwards along the shore of the Gulf of Mexico which borders the southern United States, the Central American ports on the Pacific side suffering seldom and slightly. Epidemics have been frequent along the north coast of South America from Panama to French Guiana. From any part of its endemic focal zone or of the above-mentioned areas the disease has been conveyed at times to Europe, causing for the most part mild, short-lived, and circumscribed epidemics, chiefly in the Spanish ports, though the outbreaks which occurred in the

early parts of the nineteenth century were extensive and caused severe havoc in the southern Spanish cities and in Portugal. When yellow fever reached the northern parts of Europe or Britain it never assumed epidemic characteristics, always dying out after those who came into direct contact with the infected ships had been seized with the disease.

In its endemic zone the virus usually maintains its existence in an attenuated form, blazing out occasionally under favourable conditions into the grave type of the disease frequently seen in the epidemics which prevail in the regions to which it has been carried. A fairly continuous high temperature (70° to 75° F.), with a moist atmosphere and a low elevation along the coast or mouths of navigable rivers, and an impure ill-drained soil, are the most important conditions favourable to the development of an epidemic.

Its selection of the crowded parts of populous seaports in the vicinity of the shipping is always a most striking feature. Seldom does it pass far inland, unless when carried by ships along the banks of rivers, the propagation or transmission of the disease following the routes pursued by infected ships in maritime intercourse. Very occasionally only does an epidemic appear in an elevated region, probably owing to the rapid fluctuations of temperature commonly present in high altitudes; and the outbreaks in these cases often are suddenly cut short by the advent of the cold season, the virus being apparently destroyed by a temperature a little above freezing-point.

In its epidemic form, over-crowding, bad ventilation, filth, and imperfect sewerage are shown to be important etiological factors, since the disease assumes its most virulent form and the case-incidence is much greater in those parts of the affected seaports where these conditions prevail. In the northern hemisphere the outbreaks are confined to the late summer or autumn months, the greatest mortality being in September and the first half of October. This fact is of considerable interest and importance when considered in connection with the recent demonstration of the transmission of the disease by mosquitoes.

Yellow fever attacks individuals at all ages, men and women suffering about equally, when the greater risks of exposure in men are considered, but the mortality is higher in men. The negro race suffers much less than the white, both as regards incidence and mortality, and new-comers suffer very much more than the older residents in a district visited by an epidemic. As in other infectious diseases, alcoholism, previous debility, and excesses of all kinds, mental or physical, predispose markedly to the disease.

As a rule to which there are very few exceptions, one attack confers lifelong immunity.

Formerly the disease was maintained to be highly contagious, the mechanism of transmission being accepted as direct contact with the affected and by fomites adhering to unwashed clothing, bedding, &c., and lingering in the crevices of rooms and hulks of ships. The influence of fomites has been disproved in a very striking manner by the United States Commission in Cuba. Several individuals occupied for two months a specially constructed residence kept at a temperature of 76°, from which sunlight and fresh air were excluded; the rooms were stored with the soiled linen and bedding from yellow-fever patients, and excreta from these patients were also kept in the house. Though the soiled articles were packed and unpacked daily, the seven individuals engaged in the experiment escaped, though none of them had previously suffered from the disease. The Commission further demonstrated that the blood of yellow-fever patients when injected into healthy subjects produced an attack of the disease; they proved conclusively the correctness of the theory of Finlay, advanced many years previously, that the virus of the disease could be inoculated through the bite of a mosquito (*Culex fasciatus* or *Stegomyia fasciata*) which had previously sucked the blood of a fever-stricken patient.

Though this method of the transmission of yellow fever has been demonstrated, it remains still to be proven that it is the only way in which the disease is propagated from the healthy to the sick. Guitéras has removed one difficulty in the acceptance of its universal application by emphasising the fact that the disease exists in a very mild or latent form in the native children living in an affected district, these subjects when bitten supplying to the mosquito the necessary infectivity when no yellow-fever cases are present. The remarkably successful experiment of eradicating yellow fever from Havana in 1901 by a crusade against the mosquito is accepted as a proof of the above; though it must be remembered, as has been pointed out by Sanarelli, that in the same year the disease spontaneously disappeared from Santos in Brazil.

J. W. W. Stephens summarises the facts obtained during the experimental inoculations with *Stegomyia fasciata*. The mosquito, in order to transmit the infection, must have sucked the blood of a patient neither earlier nor later than some time during the first three days of the fever. The insect can only convey the disease after an incubation-period in its own body of at least twelve days, and it remains infective for a further period of forty-five days—i.e. up to fifty-seven days after sucking the infecting blood. The young insects only lay eggs after they have sucked blood, and they begin to bite within twenty-four to thirty-six hours after fecundation, and for some days they bite during the day, after which period their habits become nocturnal, biting most frequently between

5 P.M. and 2 A.M., contagion taking place between 5.30 P.M. and 7 A.M.

The period of incubation in the human subject experimentally inoculated varies from about two to five days. Manson, commenting upon the recent experimental researches elucidating the transmission of yellow fever by the bite of *Stegomyia fasciata*, claims that they fully explain, 'Firstly, the impunity with which a yellow-fever patient can be visited by a non-immune if outside the endemic area; the mosquitoes in the vicinity are not infective. Secondly, the danger of visiting the endemic area, especially at night; the mosquitoes there are infective and active. Thirdly, the discrepancy between the incubation-period, three to five days, of the disease, and the incubation-period, fourteen days and over, of an epidemic; the necessary evolution of the germ in the mosquitoes infected by the original introducing patient demanding the space of time indicated by the difference between these two periods. Fourthly, the clinging of yellow-fever infection to ships, buildings, and localities; the persistence of the germ in infected mosquitoes (*Stegomyia fasciata*), which are known to be capable of surviving for five months, and probably longer, after feeding upon blood. Fifthly, the high atmospheric temperature required for epidemic extension of yellow fever; such temperature favours the activities and propagation of the mosquito, and is probably necessary for the evolution of the germ also in the mosquito.'

The genus *Stegomyia* is easily differentiated from the *Anophelinæ* (which play a corresponding rôle in the transmission of malaria) by the showy yellow or silvery striped markings on the legs and thorax. *S. fasciata* is a thoroughly domestic species of wide distribution and breeds in small collections of surface-water, in cisterns, and household utensils where water is allowed to remain stagnant. The genus is known to include almost a score of distinct species, several of which are suspected of being capable of harbouring the parasite which causes yellow fever.

Manson points out that Asia has never been invaded by yellow fever, probably owing to the long circuitous northern or southern route between it and the endemic yellow-fever zone, but he considers that the opening up of the Central American canal may introduce the disease through the new direct and rapid trade route into the large, swarming, and filthy cities of the East, with their non-immune overcrowded populations and defective sanitary environment. A single infected *Stegomyia fasciata* conveyed alive to the shores of Asia he considers would be sufficient to set the whole of the tropical section of the Eastern hemisphere in a blaze. The specific mosquito is found in great abundance all over the tropics, and to its susceptibility to cold is almost certainly

due the short-lived nature of the small epidemics which have frequently visited Southern Europe, and occasionally in hot seasons have even reached English shores.

The practical immunity enjoyed by the natives in the yellow-fever zone already referred to upon page 1845 is explained by Guitéras as due to mild attacks of the disease in childhood, since the mosquito can obtain its supply of virus by sucking the blood of apparently healthy native children. Manson points out that if this really be the case, as soon as mosquito extermination is effected in a district the acquired immunity will be lost within the course of a single generation, in which case a fresh importation of a yellow-fever patient will find the entire native population non-immune. The history of mild epidemics in districts or isolated regions from which the disease had disappeared for many years does not support this theory, and Manson conjectures that there are two strains of the yellow-fever virus—one of great, and another of little virulence. These are mutually protective, in the same manner as the modified small-pox, after passing through the calf, is capable of affording immunity against human variola; and he suggests that the mild strain of yellow fever, if discovered, might be found to afford protection against the ordinary virulent form of the disease if introduced as a vaccine.

Bacteriology.—Though the introduction of the mosquito-factor in the transmission of the disease strongly suggests that some protozoan organism is the causal agent, no such has been demonstrated. Sanarelli denies the truth of the mosquito-transmission, and still claims that the cause of the disease is his *Bacillus icteroides*; but his contention is disputed by all recent workers in this domain. The specific organism, therefore, must be accepted as awaiting its discoverer, notwithstanding the enormous number of different microbes which have within recent years been detected in the blood, tissues, and excreta of yellow-fever patients.

Some highly interesting and important facts have nevertheless been brought to light regarding the nature of the virus which produces yellow fever. Thus, in the first instance, not only has it been demonstrated that the virus can be conveyed from the sick to the healthy by the bite of the mosquito and by the injection of the blood extracted from the yellow-fever patient in the early stage of the disease, but it is also proven that the virus is capable of multiplying itself in the blood. This latter important fact has been demonstrated by injecting a small quantity of the serum from a yellow-fever patient into a healthy individual, who, after the usual incubation-period of three days, will exhibit the disease in a typical form, and a small quantity of the serum from the second patient may be made to infect a third healthy subject. This latter fact is accepted as proof that the virus

is organised, but the result of filtration-experiments has demonstrated that it must be of extraordinary minuteness, so that Muir and Ritchie place it in the group of *ultra-microscopic organisms*.

Reed and Carroll found that the yellow-fever blood-serum was still virulent after passing it through a Berkfeld filter. This result was corroborated by the experiments made by the members of the French Commission, who also found that the serum retained its virulence after passing through a Chamberland F. filter. Though of such extraordinary minuteness, it has been demonstrated that the organisms are capable of being filtered out by the Chamberland B. filter. Owing to its ultra-microscopic dimensions, the causal factor of yellow fever has been removed from the class of ordinary bacteria and classed as belonging to the group of suspected organisms which produce the horse-sickness met with in South Africa, the foot-and-mouth disease, and the infectious pleuropneumonia of cattle. Reed and Carroll also found that the yellow-fever virus was destroyed by exposure for ten minutes to a temperature of 55° C.

It is a significant fact in this connection that Marchoux and Simond contend that they have proved that the germs of the disease are handed down to the young mosquitoes hatched from the eggs of the mother-insect after sucking the blood of a yellow-fever patient.

Symptoms.—The *incubation-period* is variable, but the great bulk of the evidence available from clinical records shows that the duration demonstrated by experimental methods holds true for the ordinary cases met with in practice—*i.e.* three or four days, though it is stated that in temperate climates it may be much longer.

The invasion is sudden, being rarely preceded by premonitory malaise or other symptoms. The first sensation of illness is usually a chilly feeling, but a smart short rigor at the onset is not uncommon; headache, backache, muscular and joint pains, with pyrexia, rapidly follow, so that the temperature may within twenty-four hours from the initial chill reach a height of 106° in a severe case.

The physiognomy of the disease at this early stage is often marked: the face is always much congested and somewhat swollen, the conjunctivæ are of a bright red, and the eyes look brilliant, though there is a suspicion of a jaundiced tint in them. The patient feels and looks ill, anxious, and restless; his pulse is much accelerated, full, and bounding; his respirations are quick and somewhat laboured, his skin hot and dry, and his tongue moist and coated with a white fur. Anorexia is complete, nausea is present, and sometimes vomiting occurs, associated with an acutely uncomfortable sensation of heat or pain in the throat and stomach; con-

stipation is the rule. The initial head- and back-ache, muscular and joint pains, continue to increase and are often agonising in their intensity. Sometimes the restlessness becomes extreme and may pass into active delirium; more frequently at this stage, and sometimes throughout the attack, a remarkable degree of mental acuteness is observed to characterise the cerebral condition.

Upon the evening of the second or beginning of the third day the eyes are usually distinctly jaundiced, and the deep yellow discoloration which gives the name to the disease spreads downwards to the entire cutaneous surface. The urine, which from the onset showed the usual changes seen in pyrexial states, now is found to be highly albuminous and may present all the characters of the urine passed in acute nephritis. If the case is a very severe one which is going to prove fatal, there may be no drop in the temperature at this stage, or only a remission of a few hours' duration; the gastric irritability increases, the occasional vomiting of clear mucous fluid gives way to the bringing up of bile, and finally to frequent emesis of dark-stained bloody fluid, which constitutes the 'black vomit'—a striking feature of the disease. This symptom is of a hæmorrhagic nature and may be associated with melæna or bleeding from other mucous surfaces, and is generally accompanied by hæmorrhages into the skin. The tongue becomes dry, hard, and brown, and thirst is intense. The pulse at this stage—about the third day—usually shows a peculiarity almost characteristic of the disease: it tends to fall in the face of a stable or steadily rising temperature, and at a later stage, even in patients who survive, it may fall to half the normal rate.

The mental condition may remain clear to the end; more commonly delirium precedes coma; hiccough, subsultus, and suppression of urine occur, and the temperature, which may have fallen to the normal, sometimes registers a hyperpyrexial range a few hours before death. The final issue in the more malignant types of the disease may occur within forty-eight hours, or less, of the initial onset.

In the majority of instances of the disease the symptoms do not continue upon the lines of the above uninterrupted downward march. About the third day the temperature, which had reached its fastigium by the end of twenty-four hours, registering 103° , 104° , or more, and having remained high with but slight remissions, now begins to fall steadily, the pulse and other symptoms, as pain, albuminuria, vomiting, &c., showing a corresponding improvement. This constitutes what is known as the *remission* or '*stage of calm*,' and may last for two or three days, after which what is called the *stage of reaction* or *secondary fever* sets in. In some cases, however, the onset of the remission-period practically

terminates the illness, the symptoms of gradual improvement not being followed by any recrudescence of fever or other unfavourable feature, and convalescence begins. More frequently the former symptoms return or increase in severity after forty-eight or more hours, and others of a more serious character are added, as black vomit, suppression of urine, hæmorrhages from various sources, delirium, coma, &c., which may terminate in death or, more rarely, in a slow lysis or crisis with profuse perspiration, ending in recovery.

In rare cases the secondary fever may continue for many days or some weeks, with marked irregularities in the chart, the clinical picture being one of profound toxæmia, like that seen in advanced typhoid fever; and the resemblance to this may be further strengthened by profuse diarrhœa, facies Hippocratica, subsultus, &c., though the jaundiced condition tends to deepen, and skin and other hæmorrhages are present.

Relapses are not infrequent, and they are liable to occur at any time during the convalescence, and usually prove fatal.

In some epidemics of the disease the type varies widely, a profuse diarrhœa setting in suddenly, with severe vomiting, the cases simulating cholera. In others an algide type predominates, the temperature being sub-normal, and the patient succumbs from profuse hæmorrhages and collapse before the development of jaundice. In all epidemics a mild type is met with amongst the severer malignant or pernicious forms in which the pyrexia is moderate, convalescence setting in after two or three days, the nature of the attack only being recognised by the prevalence of the epidemic and the presence of a faint icteroid tinge of the conjunctiva or skin; and even this is said to be often absent.

Morbid Anatomy.—There cannot be said to be any pathognomonic anatomical change observable. The skin is always deeply jaundiced, even when the icteroid tinge was scarcely discernible during life, and the petechiæ are obvious. Evidences of the hæmorrhages which had occurred during life are present in the stomach and bowels, and similar extravasations or oozings may be found in the serous membranes and in the interior of organs. The blood shows little tendency to coagulate and contains a considerable amount of urea. In the early stages of the disease during life there is always a high percentage of hæmoglobin present. The lymphatic glands are often found enlarged throughout the body.

The heart usually shows some softening from fatty degeneration, the degree of which depends upon the stage of the fever at which the patient had succumbed. The liver-changes are more obvious, though they can hardly be regarded as distinctive. The organ is of a pale yellowish-brown colour and friable; the cells are found extensively degenerated, the type being distinctly fatty, and numerous necrotic areas are

always visible. The spleen is normal in dimensions and structure. The kidneys are the seat of changes resembling those met with in acute Bright's disease, in addition to which areas of necrosis are observable; the epithelium of the convoluted tubes shows cloudy swelling in some places and in others granular or advanced fatty degeneration, and the tubes are blocked by epithelial debris, and minute hæmorrhages may be found studded throughout the organ or under its capsule.

Diagnosis.—The mild cases previously referred to can usually only be recognised as examples of the disease when an epidemic is present.

Malaria, from its geographical distribution, is the disease most likely to be mistaken for yellow fever, but the difficulty usually only occurs at the commencement of an epidemic. The chief points in the differential diagnosis are the early appearance of jaundice, hæmorrhages, and of albuminuria out of all proportion to the other symptoms, the absence of splenic enlargement and of the malarial parasites in a specimen of the blood. The malarial blood will show in the early stages of the disease a diminished hæmoglobin-percentage, whilst in yellow fever this is always high. At a later stage the black vomit and other characteristic symptoms, notably the frequent inconsistency observable in the pyrexia and pulse-rate, will prevent errors. *Dengue* or *Dandy Fever* is liable to be mistaken for yellow fever, especially as both diseases may appear in epidemic form at the same time, as instanced by Osler in the Texas epidemic of 1897. The early presence of jaundice and of profuse albuminuria is never seen in dengue; in the latter disease the joint-pains associated with the primary and secondary rashes, itching, and desquamation are characteristic.

Relapsing Fever, sometimes marked by jaundice, albuminuria, and vomiting, can only be excluded by a microscopic examination of the blood, which will show the causal spirillum. The primary pyrexial attack in relapsing fever is always longer than in yellow fever.

Prognosis.—The death-rate, like the symptoms of the disease, varies enormously in different epidemics, being sometimes as low as 8 or 10 per cent. and as high as 75 per cent. It is always much higher amongst the new-comers than amongst the old acclimatised residents in yellow-fever districts, and greater in men than in women, and the disease is very fatal amongst the victims of chronic alcoholism.

A high range of fever at the onset, and even a moderate pyrexia (103°), which does not show any remission on the third day, is grave. Abundant albuminuria, especially when suppression is threatened, is a bad sign, as is also black vomit and other hæmorrhagic signs, notably when the temperature

remains sub-normal. Such toxæmic symptoms as hiccough and subsultus usually are a presage of death, though it must always be remembered that the most unpromising and apparently hopeless case may sometimes recover. One old patient and friend of the writer was actually placed in the mortuary of a hospital in a Brazilian port, and, becoming conscious in the night, was able to find his way out, and finally recovered. As in enteric fever, everything may look hopeful, when a sudden extensive hæmorrhage appears and causes a fatal collapse.

Rarely is recovery observed to follow in cases where the thermometer registers 106° ; children very seldom succumb to the disease.

Treatment.—Prophylactic measures have been of the greatest value in checking the spread of the disease, and sometimes in stamping it out altogether, after it has remained endemic for years in a locality. The only method to be relied upon is a crusade against the mosquitoes and the extermination of their breeding-grounds. The spread of the disease would be stopped if every patient was so surrounded by netting that the insects themselves were prevented from becoming infected. In this connection, however, it must be remembered that the mosquito may remain infective for a period of fifty-seven days after sucking the blood of a yellow-fever patient; and Marchoux and Simond have proved that the virus of the disease may be transmitted to the young insects through the eggs laid by their infected mother. It is obvious that every precaution possible should be taken by the healthy to protect themselves against the bites of the insect. As already mentioned, the mosquito does not become infected unless it sucks the blood of the yellow-fever patient some time during the first three days of the disease.

The treatment of the disease, when once established, is best conducted upon the lines recognised as valuable in the management of typhoid fever, each symptom being combated by agents employed upon accepted therapeutic principles.

Early rest in bed is imperative; fever is to be controlled by hydrotherapy, hæmorrhage by chloride of calcium or full doses of tincture of perchloride of iron, vomiting by ice and small doses of cocaine or morphine, uræmia by the hot pack and pilocarpine, and prostration by alcoholic stimulation. A routine advocated by Sternberg has found favour with many practitioners in yellow-fever districts: it consists in the administration of $\frac{1}{80}$ gr. of the bichloride of mercury and 6 gr. bicarbonate of soda, dissolved in $1\frac{1}{2}$ oz. of iced water, every hour. Sternberg treated 301 cases of yellow fever in Europeans by this method with a mortality of only 7.3 per cent., and there was no death amongst 72 cases in negroes similarly treated. Many experienced physicians advocate the inauguration of all treatment by one large dose of calomel, and place

their reliance upon quinine throughout the attack. There is a consensus of opinion that purgatives (castor oil in large doses or calomel), though often markedly beneficial when administered at the beginning of the disease, are most objectionable and dangerous if given in the later stages, especially after the supervention of the black vomit and when suppression of the urine has occurred or is threatened.

Serum-therapy on the lines advocated and practised by Sanarelli has hitherto proved valueless.

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